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STUDIES ON
RENAL COLIC AND ITS TREATMENT
BY POSTERIOR SPLANCHNIC BLOCK

BY

LARS RISHOLM

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STUDIES ON RENAL COLIC AND ITS TREATMENT BY POSTERIOR SPLANCHNIC BLOCK

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Preface

Divinum est opus sedare
dolorem

HIPPOCRATES

The customary treatment of renal colic is not always satisfactory. The practitioner, satisfied that his patient is not suffering from a serious illness, prescribes analgetics and spasmolytics, sometimes with a fairly good effect; but very often the analgesia is inadequate, and the patient is then obliged to endure considerable suffering, often for a long time, until the calculus has found its way to the bladder.

This work has been intended to contribute, if possible, to a more effective treatment of renal colic, firstly by examining as carefully as possible the nature and the mechanism of the pain, and secondly by studying the value of conduction anaesthesia, in particular posterior splanchnic block, which seems to be the most effective method of treating the pain.

Studies of the physiology of pain in animals are usually of little value, and it was therefore necessary to carry out the experimental part of the work on humans. The scope of the experiments has in consequence had to be limited in certain respects.

The value of splanchnic block was studied primarily in respect of the analgetic effect, but the possibility of using such an anaesthesia for diagnostic purposes has also been considered. Attention has also been paid to the ability of posterior splanchnic block to influence normal functions of the kidney and upper urinary tract and such reflex disturbances of function as may occur in renal colic, and which may be of importance for the passage of a calculus down the ureter and for the general health of the patient.

The work has been carried out at the Department of Surgery of the University Hospital, Uppsala, with the aid of funds from the Medical Faculty of the University. I wish to thank my chief, Professor Olle Hultén, for his kind support and good advice. For valuable technical help I wish to express my indebtedness to Miss Vive Roman and to my wife Mrs. Inga Risholm. The translation into English has been made by Sten S. Bergman, M. B., Ch. B. The diagrams have been prepared by Mrs. Maj Ulfendahl.

Uppsala, January 1954.

Lars Risholm

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Part I · Experiments on the Mechanism of Renal Colic

Chapter I

Introduction and Survey of the Literature

The attack of renal colic from a calculus in the ureter is a well-known clinical picture, with a type of pain which is usually characteristic and easy to recognize; but the mechanism of its causation is still uncertain. It has not been clearly demonstrated by what mechanism the typical colicky pain is produced, nor from which part of the urinary tract it arises. The diversity of opinions with respect to this matter is shown in Table I, where the several theories explaining the mechanism of renal colic are represented.

Two or more combined causes are indicated by some authors. It is characteristic of the prevailing uncertainty in the matter that Deuticke (1951) entirely denies the existence of hyperperistalsis of the ureter above the concrement, while others (Fedoroff 1923, Gottstein 1927, Papin 1929, Brandesky 1934) ascribe to this the greatest importance in the production of renal colic. Deuticke claims that obstruction of the ureter is associated with pronounced atony and hypomotility of the upper urinary tract. This author, in common with others (Gubergritz and Istschenko 1926, Chesterman 1945, Hoffman 1950, McCahey 1950), believes that it is principally the raised pressure above the ureteric obstruction that causes the colic, but whether the pain in that case originates in the ureter, in the renal pelvis, in the kidney substance or in the renal capsule is an open question. Local changes in the wall of the ureter (hyperaemia, oedema, ulceration, spasm), caused by a concretion, are given by some authors as contributing causes of the pain, but not much importance is attached to such changes (Gottstein 1927, Chesterman 1945, Wildbolz 1952).

The uncertainty about the mechanism of renal colic seems to be associated with our incomplete knowledge of the sensibility of the upper urinary tract. Present knowledge is derived, apart from a few investigations reported below, from clinical experience of pain occasioned by ureteric catheterisation and from incidental observations during certain roentgenological procedures.

Lennander (1902, 1906 a, b) showed in his classical work on the sensibility of the abdominal viscera that the renal pelvis was sensitive to touch and to distention,

while the fibrous capsule and the substance of the kidney were insensitive both to thermic stimuli and to traumatisation.

OCKERBLAD and CARLSON (1938) produced pain by electric stimulation of the ureteric wall by means of a specially constructed ureter catheter electrode introduced through a cystoscope. They found that the whole length of the ureter was sensitive to this type of stimulus, and were able to show how the localisation of the pain on the surface depended on the level at which the ureter was stimulated. In order to show that the pain was not due to stimulation of nerve trunks through the thin-walled ureter, the authors also used a catheter with an inflatable balloon —"the Dourmashkin balloon"—and were able, by "sharp dilatation or distention of the ureter, using 2 to 4 cc in the bulb", to produce pain of the same localisation as that produced by electric stimulation. However, it is not stated how many cases were investigated by this balloon method, nor the pressure at which the balloon was made to distend the ureter.

DOURMASHKIN (1926) himself reported, in his original work with the balloon, that distention of the lower segments of the ureter did not produce pain, but he did not indicate the pressures used.

MCLELLAN and GOODELL (1943) repeated the investigations of OCKERBLAD and CARLSON on 29 subjects and obtained the same results as these. They also examined the sensibility of the renal pelvis to faradic current and to distention and found that the pain thus produced was referred to the costovertebral angle. They were thus unable to verify the observation of LENNANDER (1906 a) that distention of the renal pelvis produced pain which in addition could radiate down to the iliac fossa and the groin. Their publication lacks important details, such as recordings of the pressure and of the strength of the faradic current.

WEENS and FLORENCE (1947), who made use of "the Dourmashkin balloon" in nephrography, reported that gentle dilatation of the rubber bag with 0.5 ml of mercury in the proximal or mid-ureter "frequently was accompanied by a very transitory pain in the flank", but they did not measure the pressure by which the balloon was dilated.

RAY and NEILL (1947) tested the pain sensibility of the upper urinary tract in their important investigation of abdominal visceral sensation before and after lumbar sympathectomy. In the unsympathectomised subjects mechanical irritation (stretching and pressure) and faradic stimulation (3 volts) of the ureter and the pelvis caused pain, but faradic stimulation of the renal capsule and pressure on the kidney at points not bordering on the renal pelvis did not do so. However, they did not use balloon distention as their pain stimulus in the upper urinary tract, as they did in the investigation of the digestive tract.

ROSS (1952) in a work on pyelo-renal backflow made some observations on the pain caused by distention of the pelvis. He found the threshold for pressure pain very divergent in different people. He explained the great variation by a condition of painful pelvic spasm occurring in some cases but not in others. There were

many subjects in his series with distinct pain but without any signs of pyelo-renal backflow, and consequently he did not believe this phenomenon to be of importance for the origin of the distention pain, as MASON (1914) had earlier suggested (see also p. 30).

TABLE I. *Theories by various authors explaining renal colic.*

Distention of the renal pelvis	Distention of the ureter	Raised intrarenal pressure with distention of the renal capsule	Hyperperistalsis in the renal pelvis and ureter above the calculus	Local changes in the ureteric wall (hyperaemia, oedema, ulceration, spasm)
			FEDOROFF (1923)	
GUBERGRITZ & ISTSCHENKO (1926)				
		GOTTSTEIN (1927)	GOTTSTEIN (1927)	GOTTSTEIN (1927)
			PAPIN (1929)	
		BRANDESKY (1934)	BRANDESKY (1934)	
CHESTERMAN (1945)	CHESTERMAN (1945)	CHESTERMAN (1945)		CHESTERMAN (1945)
HOFFMAN (1950)				
MCCAHEY (1950)	MCCAHEY (1950)			
DEUTICKE (1951)	DEUTICKE? (1951)	DEUTICKE? (1951)		
		WILDBOLZ (1952)	WILDBOLZ (1952)	WILDBOLZ (1952)

KÖHLER (1953) in his book on backflow in retrograde pyelography reported that most subjects had a certain feeling of tension in the renal region when the pressure in the pelvis was raised to between 40 and 50 mm of mercury, and that this feeling of discomfort generally passed into a distinct feeling of pain at 80 mm. Three subjects, however, did not feel any discomfort at all, although the pressure was raised to as high a level as 200 mm of mercury. KÖHLER's investigation, however, was mainly directed towards other problems, and thus his observations on pain were not very close. Furthermore, like Ross, he did not measure the actual intrapelvic pressure but only calculated it from the injection pressure at the exterior end of the ureteral

catheter, and both of them used common catheters, without sufficient control that no fluid could leak from the pelvis down the ureter outside the catheters. These facts make their figures of the pain level of the pelvis very unreliable.

The above investigations are, therefore, incomplete and usually have the weakness that unphysiological stimuli have been used to provoke pain from the renal pelvis and ureter. In the work reported below the sensibility of the ureter and the renal pelvis has been investigated more thoroughly.

In order to reproduce, as far as possible, the conditions in renal colic with a calculus within the ureter, it was proposed to produce an artificial blockage of the ureter, and to study the painful effect of the spontaneous rise in pressure above it. However, I was soon impressed by the fact that powerful pressure against the ureter wall by the balloon used as obstructing agent did not usually cause any pain at all. I then started investigating the possibility of producing pain not only by blockage of the ureter but also by raising the pressure at different levels of the ureter.

The method used here also made it possible to study the pressure in the upper urinary tract above an acute obstruction. As far as I know, such experiments have not previously been made on human subjects. Some of the findings seem to be of interest, not only from the aspect of pain physiology, but also because they clarify certain patho-physiological phenomena in connection with acute renal colic and other obstructions of the ureter.

CHAPTER II

Methods

The investigations were made on patients undergoing cystoscopy, usually without any pathological changes in the upper urinary tract. Intravenous urography had been performed previously in every case. No premedication or other analgetic was given in cases where sensibility was examined. With regard to anaesthesia for cystoscopy, see below.

Where pain sensibility is examined and results depend on sensations and statements of the subject, a critical attitude on the part of the investigator is necessary. This was the case particularly in this investigation, where the subjects were not volunteers in the usual sense but patients undergoing cystoscopy for some reason, an examination which to many was rather unpleasant. Some patients were distressed by the cystoscopy itself, and were, therefore, unsuitable as experimental subjects; no experiments were continued in such cases. Leading questions were painstakingly avoided. As the distinction between discomfort and pain is difficult to make, especially for visceral sensations, pain was registered in all cases where the subject announced the onset of any kind of disagreeable sensation.

A specially constructed ureteric catheter (made by A.C.M.I., New York, and somewhat modified by me) was introduced through the cystoscope. It was made of nylon, size Ch. 5, with two lumina, one opening at the tip and the other in an inflatable balloon 3 cm from the tip (Fig. 1). The catheter was marked at 5, 10, 15, 20, 25 and 27 cm from the balloon. By inflating the balloon it was possible to produce a local rise in pressure at different levels along the whole course of the ureter without simultaneously obstructing the flow of urine, and also to block the ureter while recording pressures above the obstruction

The investigation is based on a series of 69 experiments described partly in the following chapters and partly in Chapters X and XI. In about two cases out of three the experiments could not be completed on account of failure to introduce the catheter into the ureteric orifice, the balloon usually being the obstacle. Since dilatation of the orifice was not considered justifiable, the experiment was discontinued in these cases. These unsuccessful attempts are not included in the series.

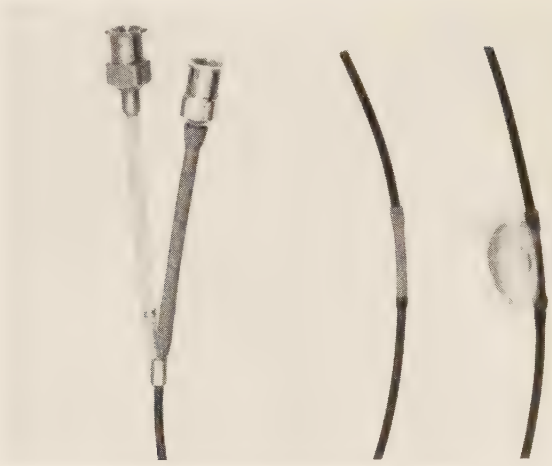

FIG. 1. Photograph of the special catheter equipped with two lumina, one of them opening at the tip, and the other into the inflatable rubber balloon.

As the technique of examining the sensibility of the ureter differs from that employed for the renal pelvis, the two methods will be described separately.

Method of examining the sensibility of the ureter

The subjects, all female, received surface anaesthesia of the urethra using "*xylocaine-gel*".

After introducing the catheter into the ureter, the balloon was filled with water to the desired pressure which was regulated with a mercury manometer (Fig. 2). In order to determine the pressure which was hereby exerted on the ureter wall, and which might be the cause of possible pain sensations, it was necessary to pay special attention to the nature of the rubber balloon. It would have been simplest to use a large balloon which in the empty state would have collapsed like a folded bag round the catheter, and which could have been filled to a diameter exceeding the maximum dilatation of the ureter without relying on its elasticity. The elasticity of the balloon wall as a factor resisting the manometer pressure could then have been disregarded. A few preliminary attempts with such folding balloons were unsuccessful, as these were too voluminous to pass through the ureteric orifice.

The only type of balloon that could be introduced was the one with which the catheter was originally equipped. This balloon was made of elastic rubber which, when deflated, tightly embraced the segment of the catheter in which one of the lumina opened.

A considerable pressure is needed to inflate such a balloon on account of its initial elasticity. But as it becomes inflated, the resistance of the rubber diminishes, so that the pressure required to maintain the balloon in an inflated state is considerably

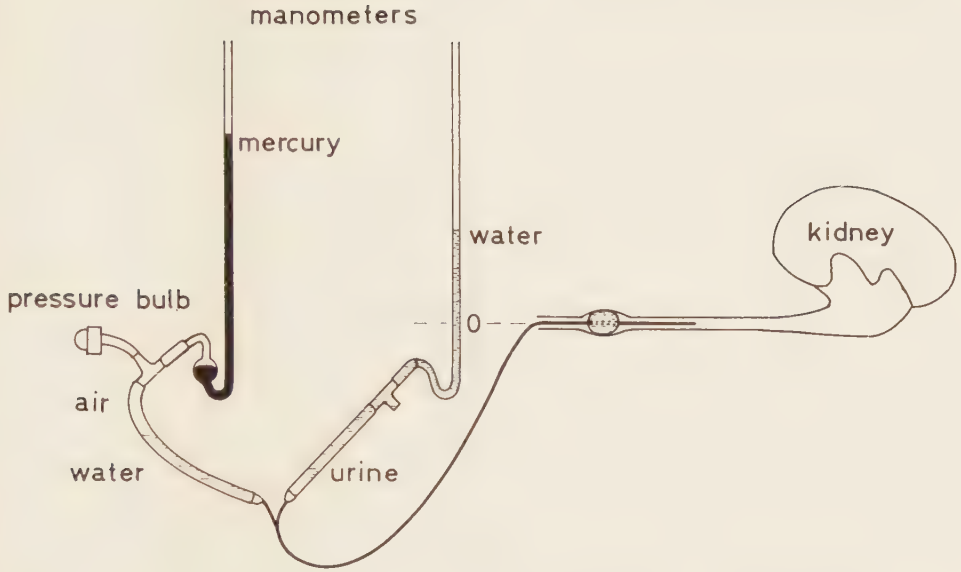

FIG. 2. The catheter is introduced into the ureter and the balloon is inflated with water to a pressure which can be read on the mercury manometer. In those cases where the sensibility of the ureter was examined, free flow of urine was permitted through the lumen opening at the tip of the catheter. Where the sensibility of the renal pelvis was examined, this lumen was connected to a water manometer, as indicated in the figure, allowing the pressure above the obstruction to be recorded.

less than that required to produce the initial inflation from the deflated state. The special properties of the balloon in this respect were defined by the following experimental model.

The catheter was passed hermetically through the rubber bung of a flask which was connected to a horizontally placed calibrated tube. A small amount of water in the latter formed a meniscus which moved as the volume of the balloon increased or decreased (Fig. 3). The reliability of this method of measuring the volume of the balloon in the flask was controlled by repeated direct measurements of the injected volumes of water. Then, by connecting the balloon to a mercury manometer and successively increasing or decreasing the pressure of the injected water, while recording the volume of the balloon on the calibrated tube, the relation between the pressure and the volume was established. The result is shown diagrammatically in Fig. 4.

To dilate the balloon the pressure required was p_1 (= 390 mm Hg; when the balloon had been used several times the pressure required was somewhat lower, but at no time lower than 360 mm Hg). At this pressure the balloon expanded to a volume of v_1 (= 2.1 ml) and at the same time the pressure fell to p_2 (= 325 mm Hg). When the pressure was further reduced, the balloon began to collapse at a pressure of p_3 (= 260–265 mm Hg), and had collapsed completely at a pressure of p_4 (= 215 mm

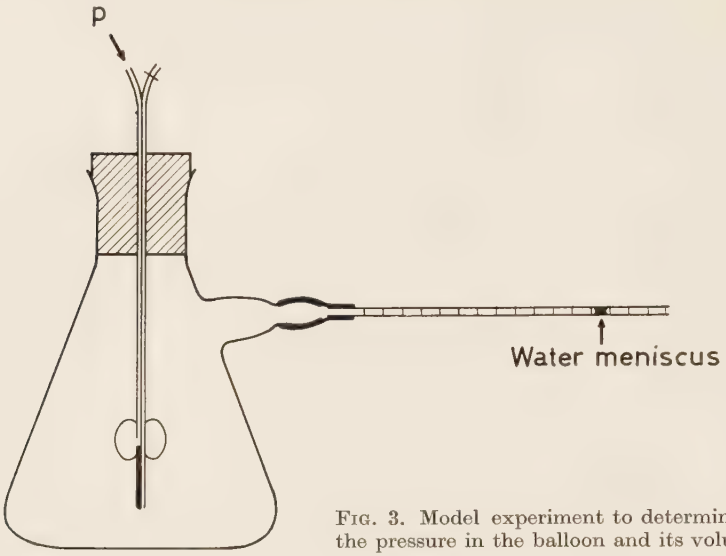

FIG. 3. Model experiment to determine the relation between the pressure in the balloon and its volume. See text.

Hg). Only at pressures exceeding p_2, as appears from the diagram, is the insufflation pressure proportional to the volume of the balloon.

In order to determine the pressure exerted by the balloon on the wall of the ureter at a given insufflation pressure, the following experiment was made (Fig. 5). The catheter was passed hermetically through the rubber bung of a flask, in which a measureable pressure could be exerted upon the outside of the balloon. The balloon was then expanded by the pressure p_1 which thereby fell to the pressure p_2. Gradually the pressure in the flask was increased until, at a pressure of p_c, the balloon began to collapse; at this pressure there was approximate equilibrium between the dilating and compressing pressures. If the pressure exerted by the elasticity of the rubber is called p_r, it follows that

$$p_c = p_2 - p_r. \tag{1}$$

The capacity of the balloon to expand within a ureter was tested on postmortem specimens. Despite the lack of tonus in such a specimen, the rigidity of the ureter wall limited the expansion of the balloon to scarcely half of the volume v_1 at the pressure p_1. The behaviour of the balloon in the ureter in vivo was followed by X-rays. The balloon had been filled with contrast medium instead of water, and it was possible to show on the X-ray photographs that the pressure p_1 actually dilated the balloon (Fig. 6). The volume of contrast medium by which the balloon was dilated was measured afterwards and it proved to be scarcely half of v_1. In order to make quite certain that the balloon actually expanded in the ureter during the pain sensibility experiments, a pressure of $p_1 + \Delta p$ was always applied. This quickly, it follows, fell to $p_2 + \Delta p$. Since the pressure of the balloon against the ureter wall,

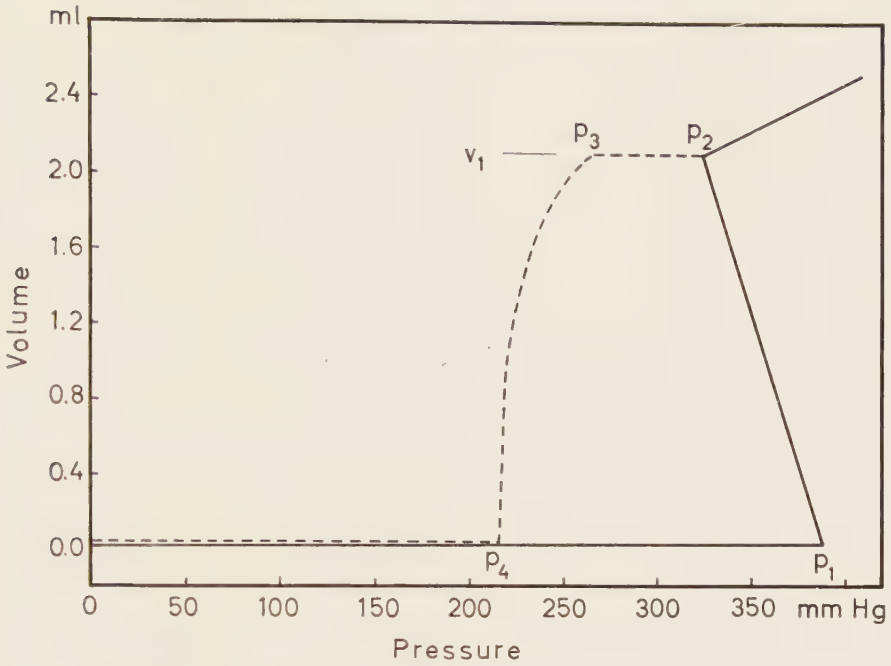

FIG. 4. Diagram showing the relation between the pressure in the balloon and its volume. The continuous line shows the relation during the dilatation of the balloon. The interrupted line shows the relation during the collapse of the balloon. See text.

x, equalled the counterpressure of the ureter wall against further expansion, the balloon being well dilated, an equilibrium existed, and this may be expressed as

$$x = p_2 + \Delta p - p_r. \tag{2}$$

Therefore

$$x = p_c + \Delta p. \tag{1, 2}$$

The pressure p_c, which in the above experiments was determined for each balloon used, varied between 110 and 130 mm Hg. A disadvantage of this method is that it allows of testing the sensibility of the ureter only at pressures greater than this. The pressure Δp was varied between 40 and 60 mm Hg and was held at this level in order to guarantee dilatation of the balloon in the ureter.

The sensibility of the ureter was examined with this method by inflating the balloon in the intramural portion, at 5, 10, 15, 20, 25 and at 27 cm above the orifice. (The length of the ureter usually varies between 27 and 30 cm.) In several cases only the lower reaches of the ureter could be examined, sometimes only the intramural portion, as the catheter could not always be made to pass higher.

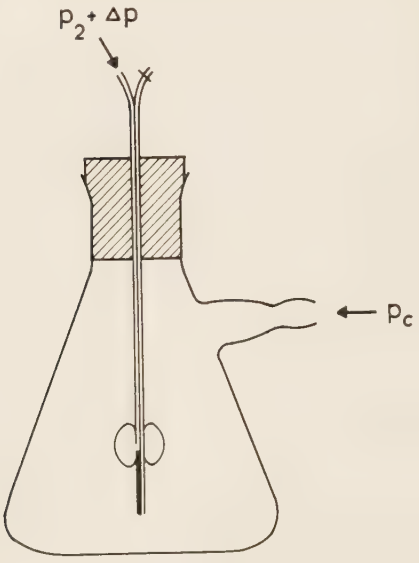

FIG. 5. Model experiment to calculate the balloon pressure against the wall of the ureter. See text.

Method of examining the sensibility of the renal pelvis

Most of the subjects were prepared for the cystoscopy with a low caudal anaesthesia (8–10 ml 1 % *xylocaine* with adrenaline 1: 100,000) which never extended higher than to S I. Four of the subjects received surface anaesthesia of the urethra with "*xylocaine-gel*". It would have been desirable to avoid conduction anaesthesia altogether and only to use surface anaesthesia in the urethra. This, however, involved certain difficulties, for it was found that reflexes, influencing diuresis, were often set in operation when a catheter was introduced into the ureter of a subject who had not received a caudal anaesthesia (see Chapter XI).

The subjects were usually made to drink 1–2 glasses of water hourly for 2–3 hours prior to the experiments, in order to ensure adequate diuresis.

The catheter was introduced into the ureter and the balloon was inflated 5–15 cm above the ureteric orifice. In a few instances, when the catheter could not be passed any further, the balloon was inflated in the intramural portion, thus blocking the ureter. The balloon was inflated to exert a pressure on the ureter wall of 150–190 mm Hg ($p_c + \Delta p$), in the same way as when testing the sensibility of the ureter. The lumen which opened at the tip of the catheter was then connected by means of a water-filled rubber tube to a graduated perpendicular glass tube which thus served as a water manometer (Fig. 2). The pressure, as indicated on the manometer, at which the subject reported a sensation of tension, pain or other feeling of discomfort was taken as the pain threshold. The onset of discomfort was usually reported clearly; the pain threshold could therefore be defined with fair accuracy.

FIG. 6. Exp. 56. X-ray photograph of the balloon filled with contrast medium and inflated 7 cm above the ureteric orifice. The pressure of the balloon against the ureteric wall was on this occasion 170 mm Hg (p_e = 110 mm, Δp = 60 mm), and the balloon contained 0.9 ml contrast medium. (The subject did not experience discomfort or pain.)

The subjects were placed supine. WÜLLENWEBER (1929) showed in the cadaver that in this position the renal pelvis is 7–8 cm lower than the pubic symphysis. I have calculated zero in this way when measuring the pressure in the pelvis. This rather approximate estimate of the level of the renal pelvis brings in an obvious source of error, but the error probably does not exceed 2–3 cm in either direction and is therefore of little consequence.

The experiments necessitated the use of a catheter of very fine lumen, and this fact brought in another source of error, namely, the difference in pressures at the renal pelvis and at the manometer. This difference depended on the rate of flow of the urine through the catheter, which was determined by means of the following experiment.

One end of the catheter was connected to a water-filled "Mariotte's flask" in which the pressure was varied, and the volume of water flowing through the catheter per minute was measured. The result appears in Fig. 7, from which the following calculations were made.

In Fig. 8 the rise in pressure above an obstruction in the ureter, as measured by the manometer, has been represented graphically by means of a diagram of typical appearance. The pain threshold was passed at the time t minutes, when the manometer showed a pressure of p cm H_2O. If a tangent to the curve is drawn through

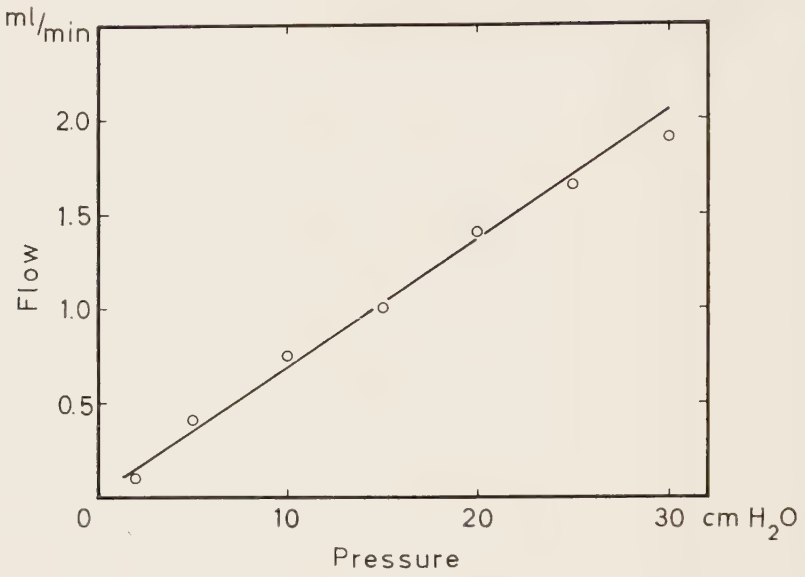

FIG. 7. Diagram showing the relation between the head of pressure and the rate of flow through the catheter.

the point (t,p) to cross the ordinate at the pressure p', it will be seen that the rate of rise in pressure at the time t was equal to $\frac{p-p'}{t}$ cm per minute. In the manometer used here 1 cm corresponded to a volume of 0.03 ml. The flow through the catheter caused by the rising pressure in the renal pelvis was therefore equal to $0.03 \cdot \frac{p-p'}{t}$ ml per minute. From the diagram (Fig. 7) may be deduced the head of pressure required to produce this rate of flow through the catheter. This head of pressure is the difference between the pressures in the renal pelvis and the manometer at the time t. The true pain threshold is thus equal to the pressure shown on the manometer when the subject indicates the onset of pain, plus the pressure which produces the rate of flow through the catheter at that moment.

The experimental error in the measured pain threshold was calculated in this way in all the experiments, but with the exception of three cases this was never more than 1 cm H_2O, and could therefore be disregarded. In experiments 36, 55 and 61, where there were vigorous diureses and relatively low pain thresholds, the differences were considerable (15, 6 and 3.5 cm H_2O respectively) and in these cases the pain thresholds were adjusted.

If an isometric manometer had been used the pressure above the ureteric obstruction would have increased more rapidly, and the pain threshold would have been passed earlier, than was the case with this method in which the obstruction came on

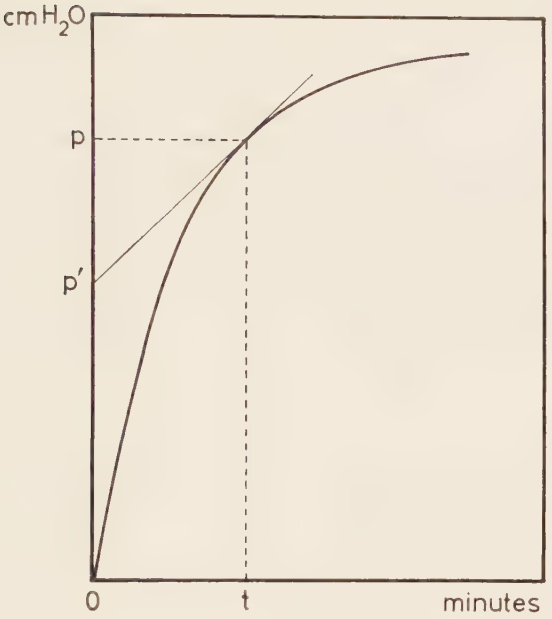

FIG. 8. Type curve showing the relation between time and rise of pressure above an artificial ureteric obstruction. The pain threshold at p cm H_2O is passed after t minutes. See text.

gradually with the rise of the fluid in the manometer. However, the volume of urine contained in the narrow manometer tube was small relative to the volume of the renal pelvis, so that there could not have been any significant retardation of the increase in pressure. Moreover, the rate of increase in pressure varied so greatly from case to case, depending on varying degrees of diuresis, that this slight time lag was insignificant.

Throughout the experiments the blood pressure was measured auscultatively with an ordinary sphygmomanometer cuff round the arm and recorded at 5 minute intervals.

Methods of studying the peristalsis above an artificial blockage of the ureter

When the sensibility of the renal pelvis was being examined, efforts were also made to study the peristalsis above the ureteric obstruction. Fluctuations in the fluid level of the manometer, apparently caused by peristalsis, could be observed in some cases, but they were inconstant and difficult to differentiate from effects of irregular breathing, speaking and movements of the subject. This method was therefore unsuitable for the recording of peristalsis. Nevertheless, in a few cases some direct observations of a certain interest could be made.

In order to get a clearer conception of the peristalsis above a ureteric obstruction, intravenous urography with serial photographs was carried out in a number of

cases. For greater detail, the intravenous urography (20–40 ml 35 % *Umbradil*) was sometimes complemented with a retrograde injection of a small quantity of contrast medium (not more than 2 ml 35 % *Umbradil*), but I preferred to avoid this for fear of interfering with the standardised experimental conditions. X-ray pictures were taken at 3–10 second intervals for 1–2 minutes. As the peristalsis normally consists of waves of contraction travelling along the ureter at a rate of 2–3 cm per second (PFEIFER 1949), with a frequency of usually 2–3 contractions per minute (TRATTNER 1932), this method should allow of at least a rough appraisal of the peristalsis. Two series of X-ray pictures were usually taken in this way, one shortly before, and the other shortly after passing the pain threshold of the renal pelvis.

CHAPTER III

Results

The result of the investigations on the sensibility of the ureter in 26 subjects is given in Table II. A pressure as high as 150–190 mm Hg exerted on the inside of the ureter in its lower segment usually produced no pain, except in the intramural portion where the innervation is different. The upper segment of the ureter was more sensitive, especially near the renal pelvis. The boundary between the lower insensitive and the upper sensitive segments of the ureter varied considerably among the subjects, but it usually lay at or about the middle of the ureter. However, in some subjects it was impossible to provoke painful sensations even in the upper segment.

The pain was characteristic in type and of fairly constant localisation. It was usually described as a definitely lateralised sensation of distention and sometimes an ache from the iliac fossa to the level of the umbilicus. With the balloon at 20, 25 and 27 cm up the ureter painful sensations were sometimes also localised to the flank and the loin somewhat below the level of the kidney. There was no other radiation of the pain.

In the intramural portion of the ureter pain could be elicited in 17 out of the 26 cases. The pain, which in 9 of these 17 cases was lateralised, was of a character quite distinct from that elicited higher up in the ureter. It was described as a sharp burning sensation in the region of the bladder and urethra.

While the ureter, as we have seen, proved to be surprisingly insensitive to stimulation by pressure, especially in the lower segment, any obstruction, followed by an increase in pressure above it, very rapidly gave rise to pain. This is exemplified by experiment 25 represented diagrammatically in Fig. 9. The pressure above the obstruction—hereinafter referred to as the pelvic pressure—rose rapidly at first, then more gradually. The pain threshold was usually reached within 15 minutes, but sometimes only after a much longer interval. Table III includes all the cases where the sensibility of the renal pelvis was examined and contains data on the rate at which the pressure increased in different cases, the pain thresholds, and the time taken in each case to reach the treshold. The active diuresis which had been encouraged in most instances, favoured a rapid rise in pelvic pressure. From the table, in

which pressures are expressed in mm Hg, it appears that the mean pressure at which the pain threshold was passed was 33 mm Hg. Minimum and maximum values for the pain threshold were 21 and 58 mm Hg respectively.

TABLE II. *Results of examination of sensibility of ureter.*

Experiment number. Subject's sex and age	Sensibility of various parts of ureter						
	Intramural part	Centimetres above ureteric orifice					
		5	10	15	20	25	27
6. f. 33	−	−	−	−	+	+	+
8. f. 62	+						
9. f. 19	−	−	−	−	+	+	
10. f. 19	−	−	−	−	−	−	+
12. f. 29	+	−	−	+	+	+	+
13. f. 47[2]	+[1]	−	−	−	−	+	+
16. f. 27	+[1]	−	−	−	−	+	
17. f. 28	+	+	+				
18. f. 62, r	+	−	−	−	−	−	−
l	+	−	−	−	−	−	−
19. f. 55, r	+[1]	−	−	−	−		
l	+[1]		−				
21. f. 48	−						
22. f. 40	+						
24. f. 29	+[1]						
26. f. 69, r	+						
l	+	−	−	−	−	−	−
28. f. 76, r	+	−	−	−			
l	+	−	−				
31. f. 49	−	?	?	+	+	+	+
44. f. 61	−	−	−	−	+		
45. f. 32	+						
46. f. 51	−						
49. f. 49	+	−	−	+	−	+	+
51. f. 49	−						
52. f. 73	−	−	+	+	+	+	
53. f. 52, r	+[1]	?	?	+	+	+	+
l	+[1]	?	−	+	+	+	+
56. f. 40	+[1]	−	−	−	+	+	
57. f. 64	+[1]		−	−	−	−	−
59. f. 43	+[1]	−	−				

\+ Pain or discomfort.
− No pain or discomfort.
? Uncertain result.
[1] No lateralisation of pain.
[2] Same subject as No. 15 (Table III).
r Right ureter.
l Left ureter.

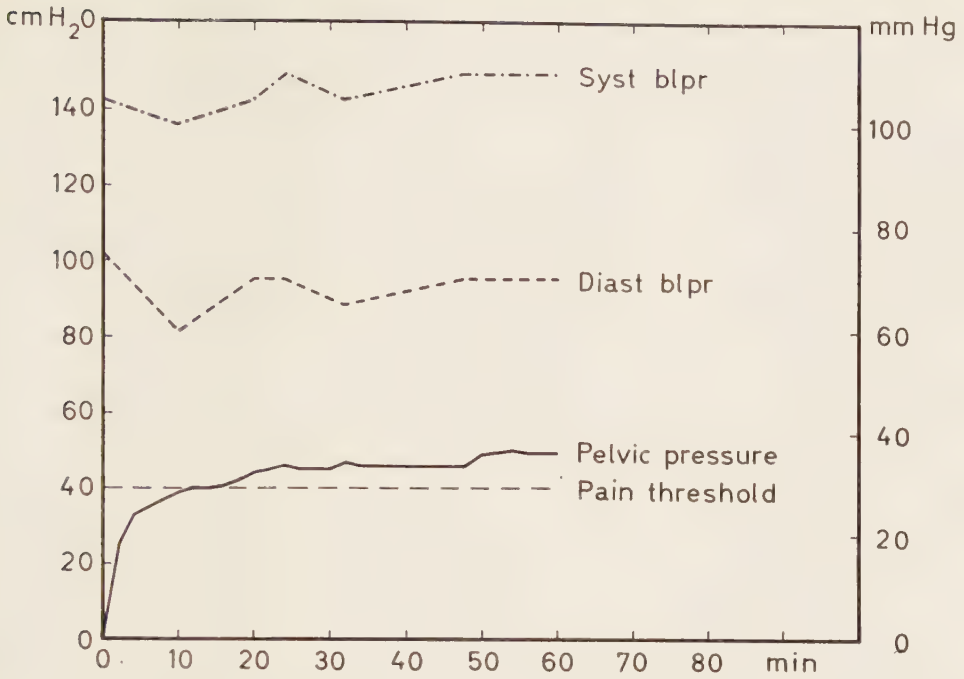

FIG. 9. Exp. 25. Graph representing the rise in pressure above the ureteric obstruction, the pain threshold, and the systolic and diastolic blood pressures.

The pain usually came on as a clearly lateralised sensation of distention in the abdomen at or about the level of the umbilicus. It rapidly took on the character of an ache, and after a further increase in the pressure of a few cm H_2O, the patient usually felt pain in the flank and loin. In a few subjects the pain started in the back, rapidly spreading to the abdomen. Two subjects (exp. 23 and 29), who had experience of renal colic from ureteric calculi, regarded the pain elicited in this way as identical. In three cases (exp. 34, 36 and 55) nausea and vomiting occurred shortly after passing the pain threshold.

Exp. 33 and 48 were carried out on one and the same person with an interval of two months; the pain threshold was practically the same on both occasions, the difference being a mere 3 cm H_2O.

When the subject was asked to bear down as much as possible, the pelvic pressure increased by 15–20 cm H_2O, but there was, naturally enough, no corresponding accentuation of the pain.

There was no noticeable change in the blood pressure in either direction with a rise in pelvic pressure.

In six of the cases where the pressure above a ureteric obstruction was studied, rhythmical fluctuations in the level of the fluid in the manometer were observed, and these appeared to have been caused by peristalsis. The fluctuations varied be-

TABLE III. *Results of examination of sensibility of renal pelvis.*

Experiment number. Subject's sex and age	Pain threshold, in mm Hg	Time taken to reach pain threshold, in minutes	Pelvic pressure in mm Hg at different times, in minutes after obstruction of ureter					
			10	20	30	40	50	60
15. f. 47[2]	30	5						
17. f. 23	29							
21. f. 48	36	11	35	38				
23. f. 39	38	10	38	40	42			
25. f. 27	29	10	29	32	33	35	36	36
29. m. 45	46	40	32	37	40	46	47	46
30. m. 21	38	14	34	39	45	48	47	
33. m. 62[1]	24	14	20	29	30	32		
34. m. 62	58	18	46	58	63	65	73	77
35. m. 77	35	8	37	44	49	52		
36. m. 73	32	1	47	55	52	52	50	
39. f. 17	30	26	15	25	36	46		
48. m. 62[1]	26	10	26	31				
55. f. 67	35	3						
61. f. 54	32	3		38	37	38	38	38
62. f. 46	25	20	18	25	35	41		
63. f. 68	21	6	29	37				
65. m. 62	28							
67. f. 18	30	4	36	32	32	32		
69. f. 45	32	5	43	49	51	52		

[1] Same subject in exp. 33 and 48
[2] Same subject as No. 13 (Table II),

tween 3 and 6 mm H_2O, and their frequency was 2–7 per minute. In four of these cases (exp. 19, 25, 30 and 61) the fluctuations decreased successively or ceased as the pressure above the obstruction increased, while in the remaining two cases (exp. 28 and 29) the peristalsis remained unaltered. In no case, therefore, could the peristalsis be shown by this method to increase with a rising pressure above the obstruction.

Attempts were made to study peristalsis radiologically in five cases (exp. 60, 61, 62, 63 and 65). In all these experiments the slightly dilated contrast-filled ureter above the obstruction appeared to be free, in its entire length, from any constriction or other alteration in contour that could be interpreted as an indication of active peristalsis. A typical series of X-ray plates is reproduced in Fig. 10. The pictures were taken at 10 second intervals in exp. 63, at a renal pelvis pressure of 46 cm H_2O seven minutes after passing the pain threshold (28 cm H_2O).

FIG. 10 (opposite). Exp. 63. Intravenous urography with a series of X-ray photographs in artificial obstruction of the ureter. The pictures were taken at 10 second intervals at a pelvic pressure of 46 cm H_2O, seven minutes after passing the pain threshold (28 cm H_2O). There are no visible irregularities in the contour of the moderately dilated ureter which could be interpreted as active peristalsis above the obstruction.

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CHAPTER IV

Discussion and Conclusions

The observation that strong distention of the lower segment of the ureter did not produce pain is surprising because it disagrees with current conceptions. WHITE, SMITHWICK and SIMEONE (1952) state that "distention of any hollow viscus is painful" (p. 127). However, they merely refer to investigations on the alimentary tract and bile ducts which have been thoroughly studied in this respect (HERTZ 1911, SCHRAGER and IVY 1928, DAVIS, POLLOCK and STONE 1932, and others). This statement would thus be inapplicable with respect to the ureter, at least in its lower segment. The observation also disagrees with the results of OCKERBLAD and CARLSON, and MCLELLAN and GOODELL, although it agrees with the incidental finding of DOURMASHKIN and with the statement of WEENS and FLORENCE that pain often, though not always, results from distention of the middle and upper segments of the ureter (p. 8).

It is difficult to explain why the ureter, unlike all other viscera, is insensitive to distention of its lower segment, and it is also difficult to explain the inconstant sensibility of its upper half. It may perhaps be reasoned that a healthy ureteric wall in itself is insensitive to pressure or distention, but that the pain sometimes elicited in the experiments was caused by distention of neighbouring sensitive tissues, such as the peritoneum. LEWIS (1943), like LENNANDER (1906 b), follows the same train of thought when he says, in referring to renal colic, that "though usually regarded as arising out of muscular contraction, it is conceivable that pains result from stimulation of nerves in surrounding connective tissue" (p. 138). The different sensibility of the different segments of the ureter, and the individual variations from case to case, would then be explained by varying relations between the ureteric wall and the peritoneum. If the pain arises from the peritoneum, one may also imagine that the variations be caused by the fact that the boundary between the sensitive parietal and the insensitive visceral peritoneum lies in the vicinity of the ureter. However, the boundary between the visceral and the somatic innervation in this zone of the peritoneum has not yet been mapped out (HOLLINSHEAD 1953). It is thus impossible to say anything about this with certainty.

The ureteric orifice differs from the rest of the ureter in respect of its innervation;

it comprises a part of the urinary bladder, and thus has sensory innervation not only from the sympathetic and parasympathetic systems but also from the somatic pudendal nerves (LEARMONTH 1931, RAY and NEILL 1947). It is, therefore, not surprising that this part is more sensitive: moreover, it is a common clinical experience that the introduction of a catheter or other instrument into the ureteric orifice often causes pain. On the other hand, instrumental manipulations in other parts of the ureter, such as attempts to dislodge calculi, are usually painless.

One weakness of the method used in this investigation is that sensitive parts of the ureter could not be tested with graded lower pressures. This would have made possible a better comparison between the sensibility in these parts of the ureter and in the renal pelvis. However, on account of the technical difficulties mentioned above, I could not find a method of carrying out the experiments in such a way.

The pain which followed obstruction of the ureter was evidently caused, not by local irritation of the ureteric wall at the point of the obstruction, but by an increase in pressure above it, the pain being clearly elicited chiefly in the renal pelvis. This pain resembled in all respects that of typical renal colic, although, for obvious reasons, the experiments had to be discontinued before the pain reached the intensity commonly found in an attack of renal colic. Vomiting, a common reflex phenomenon in renal colic, also occurred in a few cases in association with the experimentally produced pain.

It is widely believed that the pain in renal colic begins high up in the flank and gradually descends towards the groin as a result of the passage of the calculus downwards in the ureter. According to LEWIS (1943) this view is quite erroneous; the apparent descent of the pain along the course of the ureter is explained by a gradual radiation of the pain to those cutaneous segments that correspond to the innervation of the upper urinary tract, namely *Th* XI — *L* II (see Fig. 11 and Chapter V). Having carefully noted the localisation of the pain in all cases of renal colic which I examined in connection with the trial of posterior splanchnic block (see Chapter VIII), I have come to the same conclusion as LEWIS. Careful interrogation of the patient, avoiding all leading questions, hardly ever lends support to the conception that the pain descends together with the calculus. Nevertheless, it is easy to understand why many patients are quite convinced of this, for the successive segmental radiation of the pain in a protracted attack of renal colic becomes clearly associated with the downward passage of a calculus in the ureter. Such a conception may seem quite plausible, and is actually used by some practitioners to console their patients. It is only when the calculus has reached the intramural part of the ureter that pain of lower localisation, not dependent on the segmental radiation referred to above, may arise (see Chapter VIII).

It has long been the accepted view that the peristalsis above an obstructing ureteric calculus is more vigorous than normal, especially at the beginning. Such a

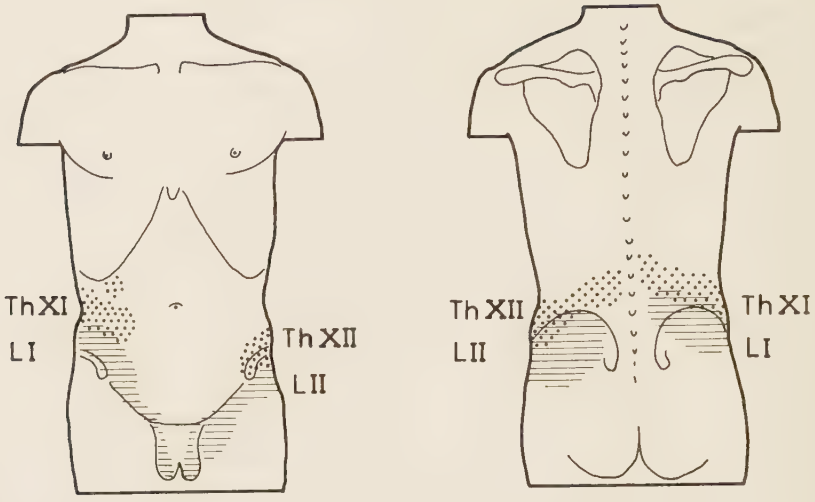

FIG. 11. Diagram showing the cutaneous segmental distribution corresponding to the visceral nerve supply of the upper urinary tract. (After KELLGREN, Clin. Sc. *4*: 35, 1939.)

conception no doubt suggests itself, with conditions then analogous to those in the digestive tract, where obstruction leads to hyperperistalsis which is sometimes adequate to overcome the obstruction. The periodic nature of renal colic, and the good effect sometimes obtained with spasmolytics, are factors supporting this conception.

However, it has never been possible, as far as I have found, to demonstrate convincingly this supposed hypermotility above an acute ureteric obstruction. On the contrary, recent observations suggest a reduced peristalsis above the obstruction. PFEIFER (1949) thus showed by means of so-called urokymography, after intravenous injection of a contrast medium, that the upper urinary tract is usually hypomotile when the ureter is obstructed by a calculus. NARATH (1951), who made extensive radiological studies on the function of the upper urinary tract, could usually observe the same phenomenon in experiments where the ureteric orifice was obstructed with a conical ureteral catheter, and a small quantity of contrast medium was injected in retrograde direction. DEUTICKE (1951), who also seems to have taken considerable interest in this question, maintained that neither hyperperistalsis nor local spasm around the concrement could be demonstrated, but that, on the contrary, both radiological and clinical observations favour hypomotility or atony of the urinary tract in renal colic.

Ordinary intravenous urography made during an attack of renal colic sometimes lends support to this concept. When the ureter is found to be filled with contrast medium as far down as the calculus, it is usually of a regular tubular shape without any contracted segments, such as one might expect to find if vigorous peristalsis were occurring.

My observations on the peristalsis, as far as the experiments allowed, were much in line with this. Even if the method gave only a very rough estimate, it can be said with certainty that the peristalsis, in contrast to the intensity of the pain, did not show any tendency to increase with increasing pelvic pressure: it rather tended to decrease. The pain produced in the experiments arose, therefore, quite independently of the peristalsis. Since there is no reason to believe that the dynamics in the urinary tract above an artificial ureteric obstruction differ from those above a calculus producing obstruction, it would seem highly probable that pain in renal colic likewise arises quite independent of hyperperistalsis as a causative factor.

The observation that the blood pressure remained unchanged with increased renal pelvis pressure is of some interest in view of the fact that data to the opposite effect have been published. GUBERGRITZ and ISTSCHENKO (1926) found in experiments with dogs that the blood pressure rose with increased renal pelvis pressure, and they regarded the phenomenon to be a result of pain. FULL (1920) interpreted similar findings in clinical experience as reflex effects. WULFF (1948), however, proved by means of experiments on dogs and cats that blood pressure did not undergo any certain changes after ligature of one ureter. He thus eliminated variations in blood pressure as a cause of disturbances of the urinary excretion in the contralateral kidney, which may occur after unilateral obstruction of the ureter (see Chapter XI).

The shape of the graph showing the pelvic pressure above an obstruction of the ureter and its relation to the pain threshold leads one to make certain reflections. Theoretically, it is correct to say that the maximal pelvic pressure equals the blood pressure in the glomeruli minus the osmotic pressure of the plasma colloids (STARLING 1899, WINTON 1934, 1937). If the mean arterial pressure is 90 mm Hg, for example, and if we assume that the blood pressure in the glomerular capillaries is at the most two-thirds of this (SMITH 1951), then, taking the osmotic pressure of the plasma colloids as 25 mm Hg, one would find that the maximal excretory pressure of the kidney would be about 35 mm Hg. In a number of earlier investigations where the maximal excretory pressure of the kidneys was determined in dogs, higher values were obtained, usually between 60 and 70 mm Hg (HERMANN 1861, HEIDENHAIN 1883, GOTTLIEB and MAGNUS 1901, HENDERSON 1905, HINMAN and LEE-BROWN 1924). The most thorough investigations in this field have been made by WINTON (1934, 1937) who found the normal maximal excretory pressure to be only about 30 mm Hg. But he showed that certain diuretics could increase this pressure up to 70 mm Hg. GUYON (1892) and HUBER (1895), also working with dogs, made the interesting observation that the pressure in the obstructed renal pelvis, having reached a maximum of 48–73 mm Hg after 1–1 ½ hours, thereupon gradually diminished.

It would have been desirable to determine the maximal excretory pressure in my experiments, but I considered it inadvisable to block the ureter for more than an hour at a time, for fear of possible risks. Also, the pain or discomfort that this would

involve to the subjects discouraged continued experiments. The shape of the pressure curves (see Fig. 9 and Tab. III), however, suggests that the pelvic pressure after one hour's obstruction approximated the maximal pressure as determined by the authors referred to above. The pain threshold had at this stage been passed in all cases, but just barely in some. And if we compare different cases (Table III), we find that the values for the pain threshold are in some cases (e.g., exp. 29 and 34) higher than the pelvic pressures in others (e.g., exp. 25, 33 and 67) after 40–60 minutes' obstruction, when, according to what has been said above, the pelvic pressures probably have very nearly reached the maximal excretory value. Since the maximal excretory pressure is apparently subject to such wide variations, it is conceivable that in some instances the pressure above a ureteric obstruction will never reach the pain threshold in a person with a relatively high pain threshold.

It seems probable that this mechanism may explain some irregularities with regard to pain in ureteric obstruction, for example the well-known inconstancy of pain in certain cases of mechanical hydronephrosis (McCahey 1950). The gradual fall of the pelvic pressure which according to Guyon and Huber begins after less than two hours' ureteric obstruction, may bring the pressure below the pain threshold fairly rapidly; this could also explain why operative ligature of the ureter does not produce pain—a phenomenon which has been considered surprising (Huggins and Scott 1945).

A calculus in the ureter, on the other hand, does not usually produce complete obstruction continuously, since leakage of urine past the calculus occurs intermittently. Renal function does not, therefore, cease as quickly as after ureteric ligature; the pressure above the obstruction is maintained by continued excretion of urine. Leakage and continued excretion cause the pressure to oscillate between values below and above the pain threshold, thus explaining the characteristic intermittency of the pain in renal colic.

Certain forms of "silent" haematuria have been thought to be caused by rupture of a renal fornix as a result of raised renal pelvis pressure in partial ureteric obstruction (Ceelen 1930, Fuchs 1931). Such a urinary obstruction could be caused by changes in the ureter itself or in surrounding structures—for example, uterine myomata, cancerous metastases, appendicitis (Hortolomei, Streya and Burghele 1935). That ruptures of renal fornices may now and then occur at such low pelvic pressures as 20 mm Hg was shown by Hinman and Lee-Brown (1924), but a higher pressure was usually required to produce fornix rupture demonstrable by pyelorenal backflow. At excretory urography backflow is sometimes seen when the renal pelvis pressure rises after application of the abdominal compressor (Ohlsson 1948) without the patient feeling any pain. A similar observation was made by Narath (1951), who injected 2–4 ml of contrast medium into the renal pelvis after obstructing the ureteric orifice with a conical catheter; he found that the type of pyelorenal backflow which is called sinous extravasation sometimes occurred before the subjects had

begun to feel pain or discomfort from the ureteric obstruction. NARATH himself, however, believes that this sinous extravasation is not caused by fornix rupture but should be regarded as "fornical absorption"; but this view is strongly opposed by several authors, e.g. by KÖHLER (1953) who is convinced that the phenomenon is caused by an actual fornix rupture.

In any case, it is clear that the renal pelvis pressure required to produce rupture of a fornix may very well be below the pain threshold, and this fact seems to favour the above explanation of these cases of "silent" haematuria.

The following *conclusions* may be drawn from the results of the investigation and the above discussion.

Marked local increase in intraureteric pressure with distention of the ureteric wall does not invariably produce pain; in the lower segment of the ureter (with exception for the intramural portion) pain can only exceptionally be produced in this way, but in the upper segment the sensibility of the ureter to pressure is greater.

An increase in renal pelvis pressure following obstruction to the flow of urine ordinarily produces pain whose character in all respects resembles typical renal colic. Although the pain threshold of the renal pelvis lies at a value usually below that of the maximal excretory pressure of the kidney, these two values are in about the same order of magnitude, and may conceivably be interchanged in exceptional cases.

The pain in ureteric obstruction is not dependent on peristalsis above the obstruction; instead of increasing, as previously believed, peristalsis very probably decreases with rising pressure above the obstruction.

Increased pressure in the renal pelvis is the essential cause of the typical pain in renal colic.

PART II · Treatment of Renal Colic by Posterior Splanchnic Block

CHAPTER V

The Innervation of the Upper Urinary Tract

In common with other abdominal viscera the upper urinary tract is innervated with motor nerves from the sympathetic and parasympathetic systems and with sensory nerves by afferent fibres which follow the motor fibres distal to the point of junction of the anterior and posterior spinal nerve roots. While the motor autonomic fibres proximal to this point run in the anterior roots, the visceral afferent fibres, like the somatic afferent, run in the posterior roots through the posterior root ganglia, where they are linked to central neurons (Fig. 12).

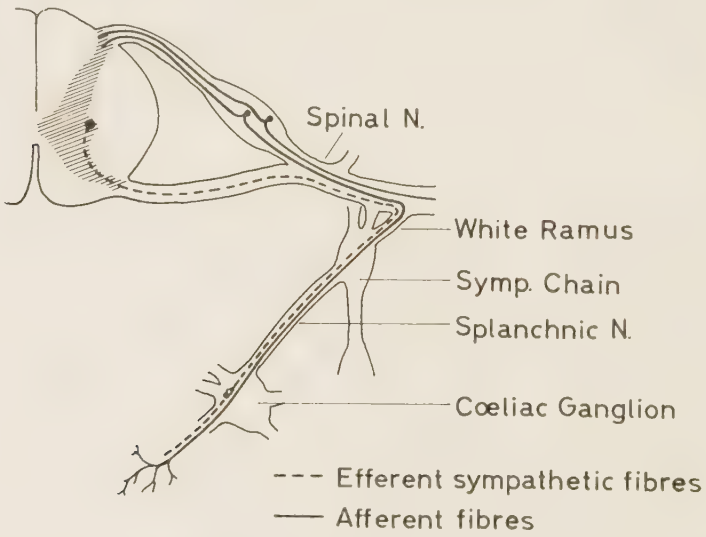

FIG. 12. The visceral afferent nerve fibres follow the autonomic motor fibres distal to the points of junction between the posterior and anterior roots. Proximal to this the visceral afferent fibres, like the somatic afferent fibres, run in the posterior roots to the posterior root ganglia where they are relayed to central neurons. (After HOLLINSHEAD, Proc. Staff Meet., Mayo Clin. *28*: 385, 1953.)

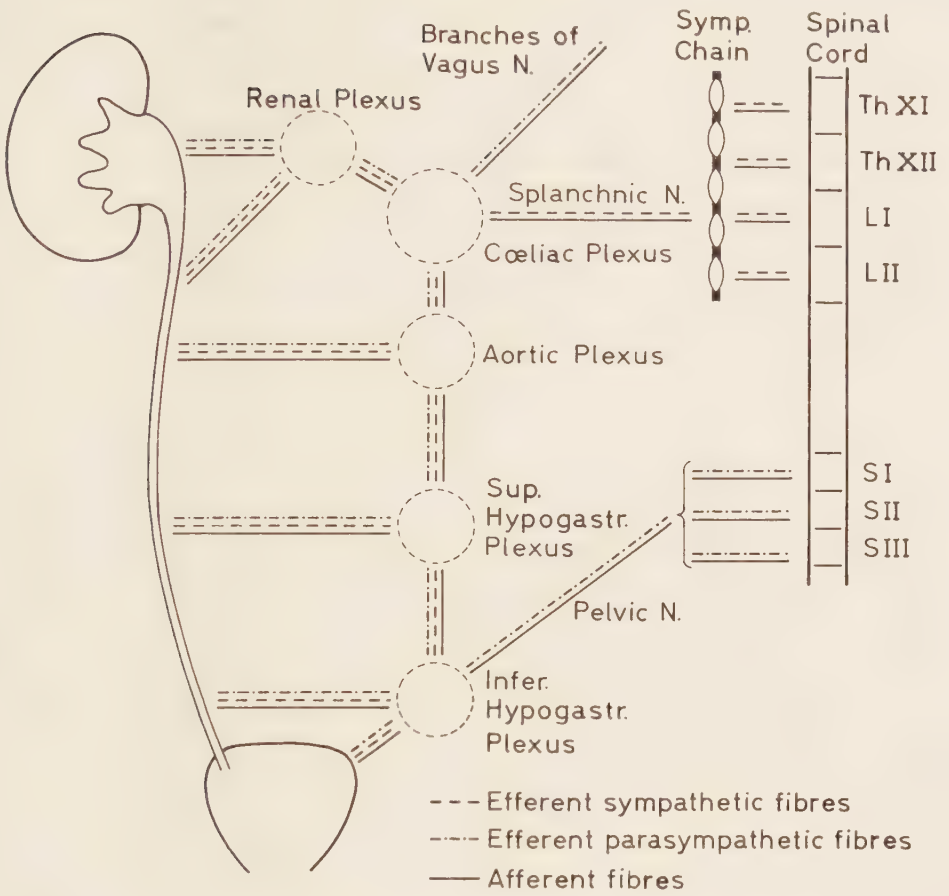

FIG. 13. Diagram showing the innervation of the upper urinary tract.

The innervation of the renal pelvis and the ureter has been mapped out by detailed anatomical studies; those of WHARTON (1932), MITCHELL (1935, 1950) and WATEL (1941) deserve mention. In Fig. 13 the innervation of the upper urinary tract as mapped out by the above authors is represented diagrammatically.

The sympathetic fibres to the renal pelvis and the ureter come from the *splanchnic nerves*, and the parasympathetic from the *vagus* and *sacral nerves*. Afferent fibres run together with these motor nerves; it should be noted, however, that no pain impulses are conducted by the vagus nerve, which conveys only certain afferent impulses from viscera, mainly those taking part in reflexes (FOERSTER 1927, GRIMSON, HESSER and KITCHIN 1947, WHITE, SMITHWICK and SIMEONE 1952).

The autonomic and the afferent visceral nerves are arranged into a network of fibres extending from the area immediately beneath the diaphragm to the region of the bladder. From the fibres of this network and from their ill-defined collections of

ganglia (shown diagrammatically in Fig. 13) fibres proceed which run to the renal pelvis, the ureter and also to the other abdominal viscera. The nerves of the renal pelvis and the upper segment of the ureter thus pass through the *coeliac, renal* and *aortic* (syn. *intermesenteric nerve*) *plexuses,* while the nerves of the middle and lower segments of the ureter first join the *superior* (syn. *presacral nerve*) and *inferior hypogastric plexuses.*

Proximal to the abdominal plexuses the pain-conducting afferent fibres from the renal pelvis and ureter run mainly in the *middle* and *inferior splanchnic nerves,* but also in the *superior splanchnic nerve,* to reach the spinal cord by the dorsal roots of segments Th XI–L II.

CHAPTER VI

Conduction Anaesthesia in Renal Colic

The good analgetic effect of conduction anaesthesia in renal colic has been known for quite a long time. Kappis (1914, 1919) paved the way for this method of treating visceral pain generally by his pioneering work on paravertebral and splanchnic anaesthesia. The method rapidly attracted interest and a considerable number of reports on its value were published, especially in Germany, for example those of Läwen (1922 a, b, 1923, 1925), Neuwirt (1922), and Lion (1927). The greatest enthusiasm was shown by those who believed that they could show that conduction anaesthesia influenced the function of the ureteric musculature in such a way as to favour the passage of a calculus (Pal 1924, Brandesky 1934 a, b). A sceptical attitude to this unproven belief (Pflaumer 1930), and the definite disadvantage that the analgetic effect was usually of short duration, were factors that gradually led to the abandonment of the method.

Conduction anaesthesia in renal colic seems, however, to have come into the foreground again in recent years, when new reports were published (Maclean, Carrol and Graves 1949, Arduini 1950, Hoffman 1950, Trevisini 1951). Access to more effective local anaesthetics with longer duration may have favoured this trend, and also the widening interest in treating severe abdominal pain of various kinds with nerve block.

Fig. 14 shows diagrammatically how it is possible to block, by means of conduction anaesthesia, nerve fibres conducting pain from the upper urinary tract, as they pass through the coeliac plexus and splanchnic nerves together with fibres from most of the other abdominal viscera. By means of a so-called *posterior splanchnic anaesthesia*, blocking of the visceral, but not the somatic, afferent fibres is achieved. A *segmental paravertebral anaesthesia*, on the other hand, blocks the spinal nerves proximal to the junction with the white communicating rami, thus interrupting both somatic and visceral afferent impulses. Paravertebral block thus also gives anaesthesia of the abdominal wall with the same segmental distribution as the visceral anaesthesia. Together with the afferent fibres, only the sympathetic of the motor fibres are blocked by the paravertebral anaesthesia. As a result of the diffuse spread

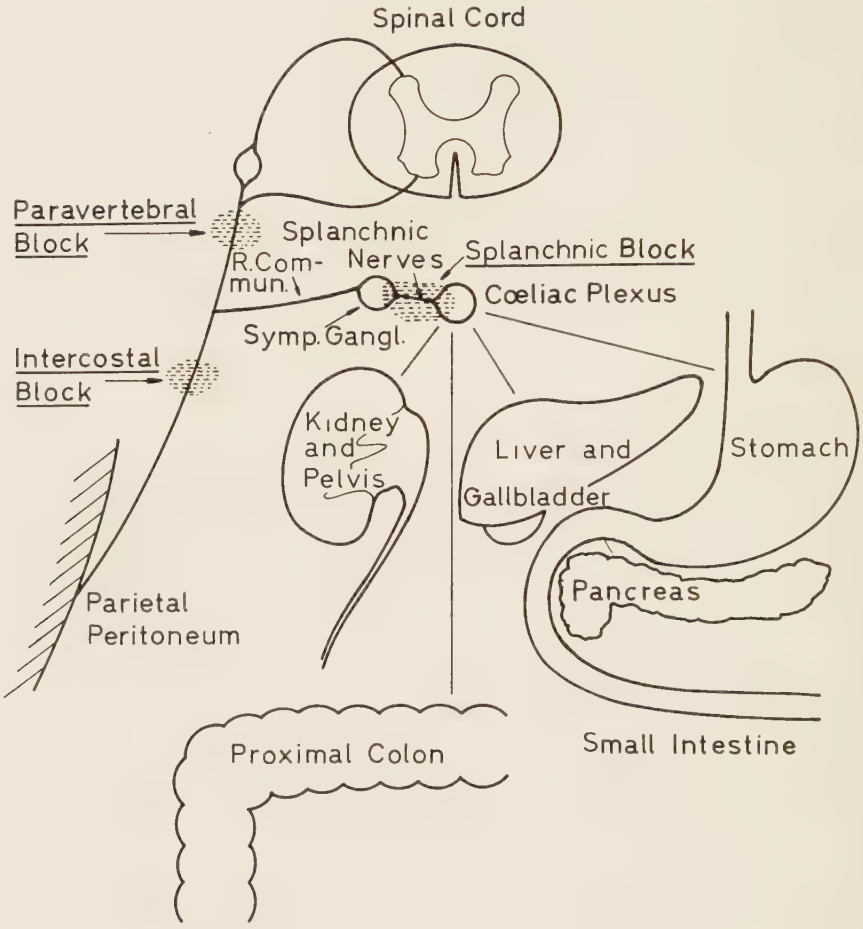

FIG. 14. Diagram of the innervation of the abdominal viscera, showing the points at which pain impulses from abdominal organs may be interrupted by conduction anaesthesia.

in the loose retroperitoneal tissues of the local anaesthetic, a splanchnic injection, on the other hand, infiltrates not only the splanchnic nerves but also a great part of the coeliac plexus, thus blocking also parasympathetic motor fibres and afferent fibres running in the vagus nerve. The term "splanchnic block" is consequently somewhat misleading, but it has been accepted generally, and is, therefore, used in this work.

After some preliminary experiments with these two types of conduction anaesthesia, both effective in renal colic, I decided to use posterior splanchnic block consistently. This type of anaesthesia has the following advantages as compared with paravertebral block:

1. It is easier to carry out and is less unpleasant for the patient, since only one injection is required.

2. Anomalies in the segmental innervation of the upper urinary tract are rendered insignificant.

3. Since afferent vagus fibres are blocked as well, vomiting and other unpleasant reflex phenomena are eliminated.

4. It offers special advantages in respect of differential diagnosis (see Chapter IX).

Paravertebral block, on the other hand, does not seem to have any advantages as compared with splanchnic block.

CHAPTER VII

Posterior Splanchnic Block

Posterior splanchnic block, originally described by KAPPIS (1914, 1919) as a complement to abdominal wall anaesthesia in abdominal surgery, and subsequently used frequently in this way (HULTÉN 1941, MACINTOSH and BRYCE-SMITH 1953), has also been of value in the treatment of different forms of visceral pain. With the development of surgery of the autonomic nervous system, splanchnic block has become an important preoperative test of the analgetic effect that may be expected from sympathectomy for e.g. inoperable malignant neoplasms and certain forms of chronic pancreatitis (RAY and NAILL 1947, DE TAKATS and WALTER 1947, DE TAKATS, WALTER and LASNER 1950). Repeated splanchnic injections with conventional local anaesthetic agents or with long-acting depot injections have been of value in such cases of intractable pain, where sympathectomy has not been performed. Splanchnic block has also been used as a preoperative test before sympathectomy for hypertension; it has also been used in certain functional disturbances of the autonomic nervous system (DE SOUSA PEREIRA 1946, LUZUY 1950, MANDL 1953). Finally it has been considerad beneficial in acute haemorrhagic pancreatitis (KLUMPP 1952).

Technique

The technique generally employed for posterior splanchnic block is still the same as that described by KAPPIS (1914, 1919, 1920), NAEGELI (1919), and LABAT (1920). The method is simple and much the same as that used for lumbar sympathetic block.

Fig. 15 shows the topography of the splanchnic nerves and the autonomic abdominal plexuses in relation to the skeleton and the kidneys. It is advisable to deposit the anaesthetic agent immediately ventro-lateral to the twelfth thoracic or first lumbar vertebra, in order to make most certain of blocking the splanchnic nerves.

The patient may conveniently be placed on his side upon a firm horizontal table, with his knees drawn up high and his back arched, in the same way as for lumbar puncture. If there is pronounced scoliosis, the anatomy is more easily appreciated with the patient sitting up, leaning forward to flex the spine.

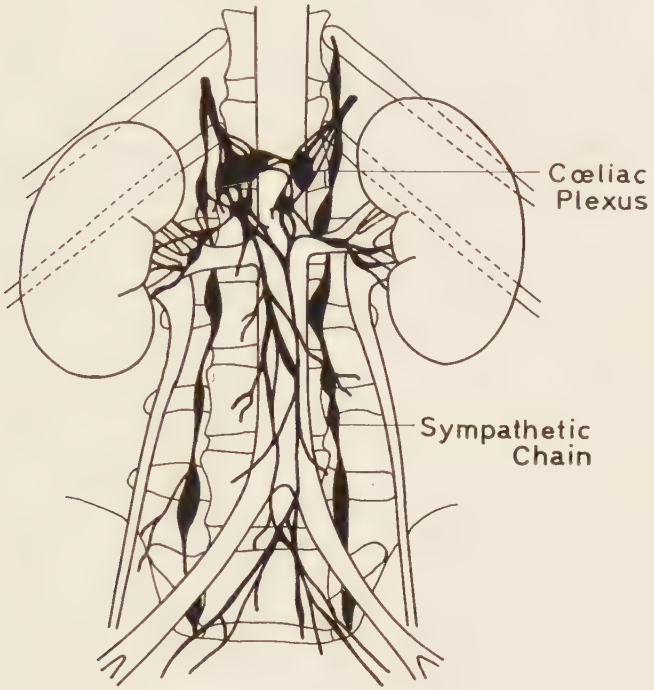

FIG. 15. Topography of the coeliac plexus, the sympathetic chain and the splanchnic nerves in relation to the skeleton and the kidneys. (After DE SOUSA PEREIRA, Arch. Surg. *53*: 32, 1946.)

The injection needle should be 12–15 cm in length and 0.9–1 mm in diameter. A finer needle is usually too supple to steer correctly at a depth.

The needle is introduced immediately below the twelfth rib, about 7 cm from the midline, and is directed 30–40° medially and 10–20° cranially (Fig. 16). At a depth of 7–8 cm the point meets a bony resistance, the dorso-lateral aspect of the vertebral body. The needle is then withdrawn a little and is redirected and advanced about 2 cm in a more sagittal direction, so that the tip of the needle glides past the vertebral body. Sometimes it is unnecessary to withdraw the needle, as it may suffice to rotate it bringing its bevelled surface medially, thus facilitating its passage past the vertebral body.

It is advantageous to introduce the needle as far laterally as indicated above. If it is introduced nearer the midline it often impinges upon the transverse process at 4–5 cm depth. It is, of course, easy to pass either above or below the same, but the patient is occasioned unnecessary pain.

Occasionally the tip of the needle comes up against a lumbar nerve, or some other nerve trunk. This gives rise to a fairly intense, radiating pain, which can be removed rapidly by immediately injecting a few ml of the anaesthetic agent. Splanchnic block can usually be given almost painlessly, if the needle is introduced carefully and if a little anaesthetic is injected continuously during the process. With a little practice the procedure can be simplified by the omission of the orientating probe onto the

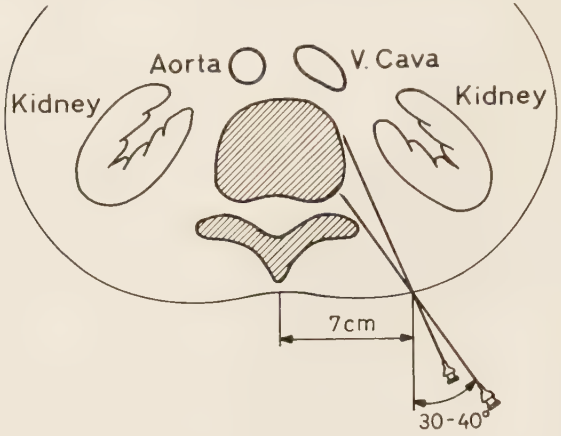

FIG. 16. The technique in posterior splanchnic block. The cannula is introduced 7 cm from the midline at an angle of 30–40° to the sagittal plane. When the point strikes against the dorso-lateral side of the vertebra, the needle is withdrawn somewhat and is then pushed in a further 2 cm at a somewhat more acute angle.

vertebral body, thereby directly introducing the needle into the correct place. Inexperienced anaesthetists are, however, recommended to use the greatest caution in order to avoid all risks.

HULTÉN (1941) pointed out the importance of injecting a large volume of the anaesthetic agent for dependable block of the splanchnic nerves, and recommended the use of 40–60 ml 0.5 per cent *procain* on each side for effective surgical anaesthesia for e.g. cholecystectomy. With the use of *xylocaine*, which spreads widely in the tissues, a considerably smaller volume is adequate. 10–20 ml ½–1 per cent *xylocaine* is thus quite sufficient for splanchnic block in renal colic.

Complications and risks

As in all other forms of local anaesthesia, accidents can occur in splanchnic block, and casualties have been reported. There are chiefly two complications which may be dangerous, namely, a *toxic reaction to the anaesthetic agent* and an *accidental spinal anaesthesia*.

The possibility of a toxic reaction to the anaesthetic agent is always present, but this risk is not greater in splanchnic block than in other forms of local anaesthesia.

If the needle is introduced too far, the aorta or vena cava is sometimes punctured. A retroperitoneal haematoma, which is of no consequence, results, and this does not constitute a contraindication to completion of the anaesthesia; the needle is withdrawn a little, to ensure that the injection will be given extravascularly. As a further safeguard it is advisable to control by aspiration repeatedly during the injection that the point of the needle does not lie in a vessel.

An accidental spinal anaesthesia may be the result if the injection needle is introduced in an incorrect manner. The risk is very small, but the following case shows that the accident can happen.

A 65-year-old man (J. No. 452/52) was to undergo cholecystectomy with posterior splanchnic block supplemented by local infiltration of the abdominal wall. The anaesthetist was not very experienced, having given splanchnic block only on a few occasions previously. 20 ml 1 per cent *xylocaine* with adrenaline had been injected on each side and the needles had barely been withdrawn when the patient suddenly became totally paralysed, ceased to breathe and lost consciousness. The pulse was fairly slow and full, and the systolic blood pressure never fell below 110 mm Hg. The patient was immediately intubated and artificial respiration under positive pressure was begun. Only after a lapse of 1 ½ hours was spontaneous breathing recovered, at first only diaphragmatically, and then gradually thoracically. Muscular motility followed, but consciousness was not regained until 3 hours after the spontaneous breathing. The patient was fully recovered on the following day, and the postponed operation was performed a week later under general anaesthesia without complications.

The reaction of the patient was interpreted as caused by a massive spinal anaesthesia following intradural injection of some of the *xylocaine*. It was not regarded as a toxic effect of the *xylocaine*, particularly in the absence of circulatory collapse and cramps.

The cause of the intradural injection in this case was most probably an inadequate knowledge of the anatomy. If the transverse process, on introduction of the needle against it, is mistakenly believed to be the vertebral body, and the anaesthetist thereupon continues with the procedure in the usual way, the point of the needle will come very close to the intervertebral foramen. If the spinal nerve is surrounded by an unusually deep root pocket following it out of the spinal canal—a state of affairs described by Rexed (1949)—then the injection can easily be given into this pocket; thus, the needle does not have to be introduced through the intervertebral foramen for an intradural injection. If the point of the needle lies in a closed fold of the root pocket, no cerebrospinal fluid will issue through the needle, either spontaneously or on apsiration. It is, therefore, necessary that the relation of the needle to the vertebral body is absolutely clear before the injection.

A less serious complication seen once in a while is *pneumothorax*, which may result if the needle passes in a more cranial plane than intended and pierces the diaphragm and pleural sinus.

The *fall in blood pressure* is usually quite insignificant, seldom more than 20 mm Hg. In unilateral splanchnic block the blood pressure is often completely unaffected.

Needless to say, it is not advisable to treat a usually benign disease, such as renal colic, with therapeutic agents that in themselves are dangerous. From reviews of splanchnic block published by de Takats (1927) and Finsterer (1936), the mortality rate has been calculated to be about one case in ten thousand (Hultén 1941). Posterior splanchnic block has been used extensively in the Department of Surgery, University Hospital, Uppsala, mainly as an anaesthetic for cholecystectomy. Since 1940 over three thousand splanchnic injections have been given without a serious complication, apart from the one described above. In view of this, I venture to state that *splanchnic block, carried out in the correct manner, is fraught with no greater risks than any other local anaesthesia, and that its use as an analgetic agent is perfectly justifiable even in benign conditions.*

CHAPTER VIII

The Analgetic Effect of Posterior Splanchnic Block in Fifty-one Cases of Renal Colic

In order to evaluate as precisely as possible the practical application of splanchnic block as an analgetic, I used the method consistently in all cases of renal colic that I had the opportunity to treat during the period January 1, 1952, to June 30, 1953. Only cases treated by myself and observed carefully with respect to the effect of the anaesthetic are included in this series, although several of my colleagues in the Department of Surgery used splanchnic block at the same time as their routine method of treating renal colic.

Of these fifty-one patients, forty-four were men and seven were women; forty-three were treated as outpatients and eight were admitted.

Clinical examination, including examination of the urine, preceded treatment. In a few cases doubt existed as to the diagnosis, and here splanchnic block proved to be of help in the differential diagnosis (see Chapter IX). The examination was always complemented with intravenous urography, usually as soon as the pain had been suppressed by splanchnic block, or—in a few instances—shortly before.

Xylocaine was used consistently on account of its considerable advantages as compared with procaine, particularly in respect of duration. In view of the danger of toxic effects I preferred not to use a *xylocaine* solution stronger than 1 per cent (with adrenaline 1:100,000), although the duration is greater with a 2 per cent solution. 10–20 ml were injected on the side of the pain; it was never given bilaterally.

The analgetic effect was excellent; all patients were very rapidly and completely relieved of the typical pain. Some patients were relieved while the injection was still in progress, and most of them within a minute. The typical percussory tenderness in the renal angle, and any abdominal tenderness, disappeared. Not only pain but also nausea, vomiting and malaise vanished immediately. Although none of the patients in this series had the severe degree of meteorism that is occasionally seen in renal colic, some of them complained of an uncomfortable abdominal distention; this was also abolished by splanchnic block. The contrast between the agonised

state before and the perfectly relieved state after the injection was often dramatic, and was greeted by the patient with expressions of pleasant surprise.

After the typical, severe pain had been relieved, seven patients reported a desire to micturate and a sensation of burning, of which they had not previously been aware, in the region of the groin and bladder. In all these cases it was shown by means of intravenous urography that the calculus was situated in close proximity to the bladder, possibly in the intramural portion of the ureter. This sensation disappeared when the splanchnic block was supplemented with a low caudal anaesthesia reaching up to S I. This observation is fully in accordance with what has been pointed out in Chapters III and IV—that the intramural portion of the ureter, having the same innervation as the bladder, is more sensitive than other parts of the ureter.

In thirteen cases the pain did not return, and calculi shortly passed per urethram, having apparently descended into the bladder while the anaesthetic still acted. In five cases pain relapsed after 24 hours or more. In the remaining thirty-three cases, pain returned usually after 2–3 hours; splanchnic block was repeated once or twice in eleven of these, while general analgetics and spasmolytics were administered to the others.

There were no complications of any kind. The blood pressure was not recorded systematically, but there were no signs of orthostatic disturbances. Those treated as outpatients were permitted to leave the hospital about thirty minutes after the injection, and were then able to go home completely symptom-free.

The analgetic effect of splanchnic block is thus complete, and the pain can be treated more effectively in this way than with general analgetic and spasmolytic drugs. Even if such preparations can often produce considerable relief of pain, they do so at a dosage which gives undesirable side-effects, especially somnolence.

An important disadvantage, however, is the comparatively short duration of splanchnic anaesthesia—although a few hours' respite with complete relief from severe pain gives valuable physical rest and mental encouragement. It is usually difficult in practice to repeat the injection more than twice or thrice; moreover, the duration of conduction anaesthesia becomes shorter every time it is repeated (Brown 1948, Risholm 1950).

It would be ideal to use a *local anaesthetic with a depot action*, but, unfortunately, there is as yet no agent with the necessary properties. The preparations which have been marketed up to the present time are somewhat irritating to the tissues, and sometimes of a rather poor anaesthetic action. I have tried such preparations in a few splanchnic injections for renal colic, but the results were generally not quite satisfactory.

In view of the intense research work which is being carried out in this field it seems reasonable to hope that it will soon be possible to choose between local anaesthetics of a few hours' duration and such of a few days' duration. In that case, the question would assume greater importance whether splanchnic block, apart from

its analgetic effect, has any effect on the functions of the kidney and ureter—factors of great importance for the passage of the calculus down the ureter. It is clear that a calculus can descend towards the bladder whilst the kidney and ureter are denervated; this occurred in several of my cases. However, as these are problems which have been discussed extensively, they will be dealt with in greater detail in a separate chapter (Chapter X).

CHAPTER IX

The Diagnostic Value of Posterior Splanchnic Block

The idea of using conduction anaesthesia for diagnostic purposes in acute abdominal conditions soon developed out of its use as a method of treating abdominal pain.

By selectively blocking different segments by means of *paravertebral anaesthesia* it sometimes became possible to differentiate between pain arising in different viscera, e.g. the gallbladder and the appendix (Läwen 1922 a, b, 1923, 1925, Kappis and Gerlach 1923, Kulenkampff 1923). This method is, however, unreliable in many ways, especially on account of the spread of the disease process onto neighbouring segmental areas. Moreover, it does not give any information where the organs in question are of the same segmental innervation, e.g. the urinary tract and the appendix. This method has fallen into disuse, having no application in acute abdominal conditions.

Splanchnic block, on the other hand, is of greater practical application as a diagnostic aid. As mentioned previously, such an anaesthesia *merely blocks the visceral afferent nerve tracts, leaving the somatic fibres intact* (Fig. 14), *and the innervation of the sensitive parietal paritoneum is thus not affected*. Only purely visceral pain can, therefore, by eliminated by splanchnic block; in disease processes where the parietal peritoneum is irritated, i.e. in most serious acute abdominal conditions, the pain remains to a greater or lesser degree.

The kidneys and the upper urinary tract, as well as parts of the ascending and descending colon, receive *unilateral* unpaired innervation; all other abdominal viscera have *bilateral* innervation (Burton-Opitz 1916, Ray and Neill 1947, Bingham, Ingelfinger and Smithwick 1950). By virtue of this fact, unilateral splanchnic block can sometimes be used to differentiate between an upper urinary tract affection and such conditions as *Cholelithiasis*. The value of this method, however, is limited by the fact that a splanchnic anaesthesia intended to be unilateral gradually becomes bilateral on account of diffusion of the anaesthetic agent across the midline.

With a thorough knowledge of the innervation of the abdominal viscera it may thus be possible to use conduction anaesthesia for diagnostic purposes; but in *acute*

abdominal conditions the only practical method is splanchnic block, which is of value when a purely visceral disease is to be distinguished from one which by peritoneal irritation gives rise to pain impulses conducted also along somatic afferent fibres.

In order to submit this argument to the trial of practice, I gave splanchnic anaesthesia in a large number of acute abdominal conditions, observing the analgetic effect. A discussion of the results would to a certain extent go beyond the scope of this study. It suffices, therefore, to report the effect of splanchnic block in *appendicitis*, for it is a common diagnostic problem to differentiate between appendicitis and rightsided renal colic.

Thirty-one patients with acute appendicitis or suspected appendicitis received splanchnic block immediately before appendicectomy was performed. In order to render the test more exacting, I chose predominantly patients with mild symptoms; this explains why more than half of the cases were found at operation to have healthy appendices but signs of acute *enteritis* or *mesenteric lymphadenitis*. All the patients had tenderness at McBurney's point and in addition spontaneous pain of greater or lesser severity. In seventeen cases unilateral splanchnic block was given, and in fourteen cases bilateral, with 20–25 ml ½ or 1 per cent *xylocaine* (with adrenaline 1 : 100,000) on each side.

The results appear in Table IV. *All the patients had residual symptoms after the injection; the spontaneous pain commonly disappeared or diminished, but the tenderness remained unchanged.*

It need not be surprising that the symptoms remained in the eight cases where the appendix was grossly inflamed and where there were signs of irritation of the parietal peritoneum. But it is remarkable that the symptoms did not clear up in the seven cases of mild appendicitis, and even more so in the sixteen cases where the appendix was healthy, and merely a mild enteritis with or without mesenteric adenitis was present.

The visceral afferent fibres from the appendix and distal ileum reach the 11th and 12th segments of the spinal cord via the coeliac plexus and splanchnic nerves (PITKIN 1946) and are, therefore, blocked by a splanchnic anaesthesia. Since not even bilateral splanchnic block eliminated the pain in these cases, the painful stimuli must have been conducted at least in part by somatic afferent fibres.

According to LENNANDER's (1902, 1906 b) conception of the sensibility of abdominal viscera, now refuted on certain points, the pain in appendicitis is caused by spread of the inflammation via the lymphatics—mainly to the highly sensitive subserous tissues of the posterior abdominal wall which is innervated by somatic nerves. Nobody denies that the pain of appendicitis and other inflammatory diseases of the intestine may *partly* be caused in this way, although LENNANDER's belief that *all* pain in diseased conditions of the abdominal organs arises extraviscerally is now disproved.

It seems probable that the pain and tenderness which remained after splanchnic block in the cases of appendicitis and enteritis can be explained by LENNANDER's

hypothesis. It may be conceivable, too, that an irritation of the parietal peritoneum, sufficient to produce pain, exists even in mild cases of appendicitis and enteritis, where signs of peritonitis cannot be shown without histological examination.

TABLE IV. *Analgetic effect of splanchnic block in renal colic and presumed appendicitis.*

Condition	Total number of cases receiving splanchnic block	Number of cases receiving unilateral splanchnic block	Number of cases receiving bilateral splanchnic block	Analgetic effect of splanchnic block
Presumed appendicitis immediately before appendectomy	31	17	14	
Acute appendicitis with obvious peritoneal irritation	8	4	4	No freedom from pain or tenderness
Acute appendicitis without obvious peritoneal irritation	7	6	1	
Acute enteritis or mesenteric lymphadenitis	16	7	9	
Renal colic	51	51	—	Complete freedom from pain

A similar situation arises if a urinary tract affection spreads into the surrounding retroperitoneal tissues, so that a pain component, conducted by somatic fibres, is superadded. *Pyelitis* is usually limited to the renal pelvis and the adjacent parts of the renal parenchyma, but a surrounding lymphangitis and perinephritic oedema often develop. This explains the inconstant effect of splanchnic block in pyelitis. Thus, in five cases of typical pyelitis, where splanchnic block was tried, two became entirely free from pain while the remaining three retained pain and tenderness. One of these cases is particularly instructive.

A 48-year old man (J. No. 1/53), paraplegic for the last 1½ years as a result of a spinal fracture involving Th XII, L I and L IV, became ill with an attack of rightsided pyelitis accompanied by rigors and pyrexia. On account of severe pain, resembling renal colic, a rightsided splanchnic anaesthesia was given, using 20 ml 1 per cent *xylocaine* (with adrenaline 1:100,000), whereupon pain and tenderness disappeared completely. As it was queried whether a ureteric calculus was the cause of the severe pain—the patient having previously had urinary calculi—an intravenous urography was performed forthwith; this showed normal urinary excretion without evidence of calculi. The pain returned gradually and after six hours a new splanchnic block was given, using an

identical technique. The effect was smaller this time, insofar as the pain and tenderness persisted to some extent. The pyelitis then ran the usual course with a gradual fall in the temperature.

The good effect of the first splanchnic block may be explained by the fact that the inflammation at that time was limited to the renal pelvis. A few hours later the inflammation had begun to invade the lymphatics and the surrounding tissues innervated by somatic afferent fibres which were unaffected by the second splanchnic block.

In summary it can be said that *the pain in renal colic differs from that of appendicitis—and certain other, diagnostically often difficult acute abdominal diseases—insofar as the pain of renal colic disappears completely after a unilateral splanchnic block while the pain of appendicitis persists.* Intravenous urography is presumably still the surest method of differentiating between renal colic and e.g. appendicitis, but it is a somewhat cumbersome procedure for which there may be no available facilities. *Splanchnic block is, therefore, a valuable complement to other methods of examination in obscure acute abdominal conditions where a ureteric calculus is suspected.*

CHAPTER X

The Effect of Posterior Splanchnic Block on the Normal Functions of the Kidney and the Ureter

An effective ureteric peristalsis and a brisk urinary flow are factors which favour the passage of a ureteric calculus. The extent to which splanchnic block influences the normal functions of the kidney and ureter is therefore of some interest. In the following discussion I have disregarded the effects of splanchnic block on reflex disturbances of renal function which may occur in cases of renal colic, as I will deal with these effects more fully in the next chapter.

The effect on ureteric peristalsis

The function of the ureter under normal and pathological conditions is still in many respects unclear on account of difficulties in recording peristalsis. The following methods have been used:

1. Examination of excised ureters stored in Ringer's solution.
2. Kymographic recordings by suspension of a segment of a ureter in situ.
3. Recording of variations in intraureteric pressure by means of a catheter in its lumen.
4. Recording of action potentials of the ureter.
5. Radiological examination of the motility of the ureter (serial photography, roentgencinematography and so-called urokymography).

Apart from these methods of recording peristalsis, there are opportunities to make direct visual observations; of the dissected ureter; of the jets of urine at the ureteric orifices, i.e. "chromocystoscopy"; and finally, by X-ray screening.

All these methods have contributed to our present conception of the function of the ureter and have been used to determine the role of the innervation in the peristalsis. Though all the methods have certain failings, methods 3–5 are to a certain extent practicable for studying ureteric peristalsis in human subjects. The radiological method, with intravenous injection of contrast medium, is the most physiological among these, since the other two necessitate the introduction of a recording instru-

ment into the ureter, a procedure which in itself may produce functional disturbances.

The question whether ureteric peristalsis is dependent on the innervation has been a matter of lively debate ever since ENGELMANN (1869) stated that the ureter acted altogether autonomously. GRUBER (1933) has thoroughly reviewed the earlier publications in this field.

Of the more recent investigations, that of LAPIDES (1948) deserves mention. He employed TRATTNER's (1932) method of recording the peristalsis in man, using a catheter introduced into the ureter. The investigation showed that the peristalsis was unaffected by blocking of the autonomic ganglia with tetraethylammonium, by a high spinal anaesthesia, by curare or by several other drugs. He concluded that "tonus and rhythmic contraction of the intact human ureter are entirely independent of the central nervous system including the autonomic nervous system and all its ganglia".

LICHTENAUER (1948) came to a similar result in his work on dogs and cats where he studied the peristalsis radiologically after excising various portions of the autonomic nervous system. He believed, admittedly, that the autonomic nervous system exercised a superior control over ureteric peristalsis, but that autonomous activity, quite adequate for normal function, occurred even when such control was interrupted.

The view of LAPIDES, in essentials shared by LICHTENAUER, seems to be well documented, and is at present generally accepted; *there is, therefore, nothing to suggest that one would influence the ureteric peristalsis appreciably by a splanchnic block* (see p. 35).

The effect on urinary excretion

Ever since CLAUDE BERNARD (1859) proved that section of the splanchnic nerves increased the urinary flow, a large number of articles have been published concerning nervous influence on renal function. Most of the investigations were made before the modern conception of renal function had been accepted, and have, therefore, no longer the same interest. They are reviewed by e.g. BIETER (1930), CALDWELL, MARX and ROWNTREE (1931), and RHOADS, VAN SLYKE, HILLER and ALVING (1934), and will not be discussed here.

The most reliable methods whereby the function of the denervated kidney has been studied are those in which transplantation of the kidney has been achieved. Complete denervation of the kidney is guaranteed only when the kidney has been removed out of the body and then implanted again, preferably in a new situation. As regards the urinary flow in such a kidney, there is usually a tendency towards polyuria with diminished power of concentration, but otherwise nothing of importance to the present discussion (LOBENHOFFER 1913, QUINBY 1916, DEMPSTER and JOEKES 1953).

In order to verify these conclusions, I studied the effect of splanchnic block on

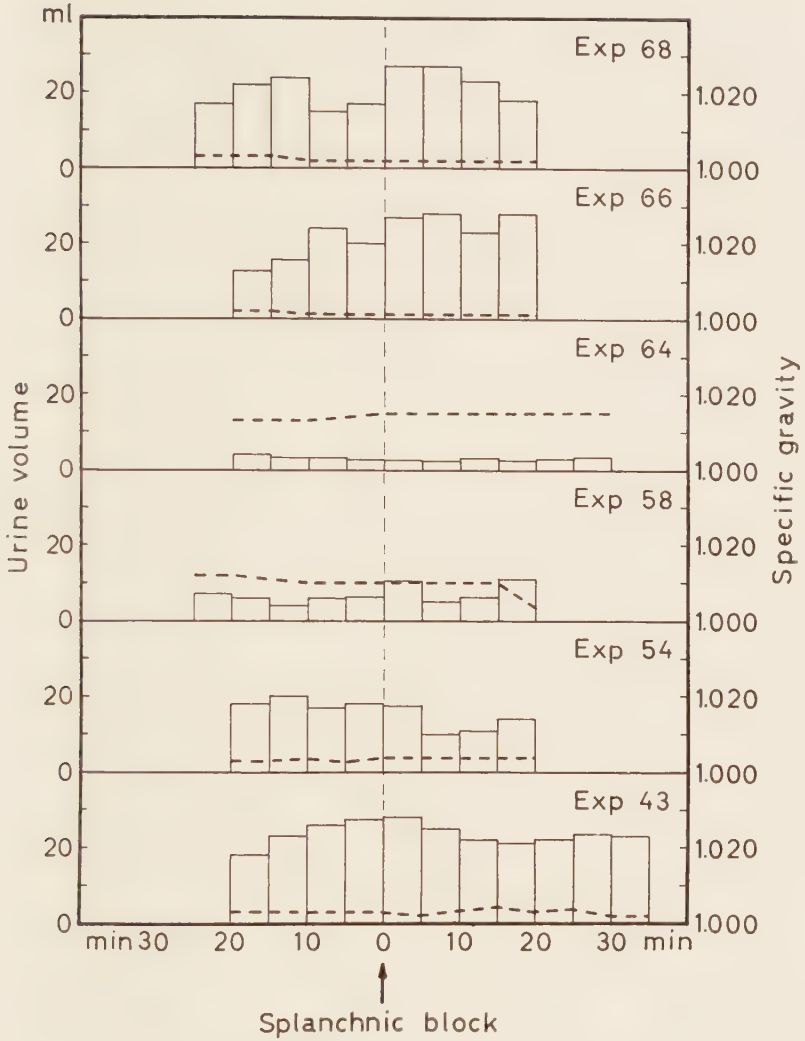

FIG. 17. Diagrams showing urinary excretion and specific gravity of the urine before (to the left) and after (to the right) splanchnic block in six experiments. The columns indicate urine volumes and the interrupted line specific gravity.

urinary flow in a series of experiments on human subjects. The technique of the splanchnic block was the same as that used in the treatment of renal colic. The details of the experiments were the same as those described in Chapter II, except that the balloon catheter had been replaced with an ordinary ureteric catheter, size Ch. 6, having ten holes spaced at 1 cm intervals from the tip. This catheter was introduced into the ureter. A low caudal anaesthesia was given before cystoscopy in order that reflexes from the bladder and ureteric orifices might be prevented from

influencing the urinary flow (see Chapter XI). An intravenous injection of indigo-carmine, which imparted a deep blue colour to the urine, made it possible to observe through the cystoscope that there was no leakage of urine alongside the catheter. The urinary flow was then measured for each five-minute period, and the specific gravity of each specimen of urine was determined by weighing an exact volume in a small measuring flask.

When the urinary volume and specific gravity had been followed for 20–30 minutes a splanchnic block was given, using 20 ml 1 per cent *xylocaine* without adrenaline. The urinary volume and specific gravity was thereupon followed at least another 20 minutes.

As expected, *splanchnic block did not have any significant influence on the urinary flow*, neither in the cases with a brisk diuresis, nor in those whose flow was somewhat less (Fig. 17).

To summarise: splanchnic block does not in normal circumstances affect the function of the ureter and kidney in such a manner as to influence the passage of a ureteric calculus.

CHAPTER XI

The Effect of Posterior Splanchnic Block on Reflex Disturbances of Urinary Excretion in Renal Colic

Before entering on the question of possible effects of splanchnic block on reflex disturbances of urinary output in renal colic, it is necessary to discuss the existence of such reflexes. The literature in this field is profuse and opinions differ on fundamental points. The various conceptions are based partly on clinical observations and partly on extensive animal experiments. Experiments on human subjects, on the other hand, have very seldom been done, which is a shortcoming, because reflex irritability varies greatly from species to species. During the course of the investigation described in Part I there were opportunities to study reflex disturbances of urinary flow. It was impossible, for practical reasons, to study these reflexes so systematically and so extensively in human experiments that anything could be proved by them. But some of the observations are relevant to this discussion.

Ipsilateral disturbances of urinary excretion

The pressure relations above an artificial obstruction of the ureter suggest that urinary excretion usually proceeds until the pressure in the renal pelvis prevents further glomerular filtration (see p. 29); it is, therefore, not a question of a reflex mechanism halting the urinary output (Wulff 1941). This belief is upheld by the finding that denervation of the kidney in an experimental animal does not affect its maximal excretion pressure (Winton 1937). However, in some cases with ureteric obstruction one has found reno-vascular reactions, probably reflex in nature, which are able to influence urinary excretion. Trevisini (1952), who carried out aortographies in renal colic, noticed that the renal artery on the affected side was often contracted; also, Bianchi and Dell'Adami (1950) found that ligature of one ureter in experimental animals resulted in an ipsilateral spastic contraction of the renal artery, demonstrable by aortography.

The significance of this vasoconstriction, as far as function is concerned, is still

uncertain but, theoretically, a decreased filtration pressure and a decreased urinary output should result.

In three cases, where renal pelvis sensibility was being examined, I gave splanchnic block at a time when the pelvic pressure was judged to approach the maximum (in exp. 29, fifty minutes; in exp. 30, thirty-three minutes; in exp. 69, thirty minutes after obstructing the ureter). The denervation was effective, as judged by the fact that the pain disappeared immediately. As expected, the splanchnic block did not produce any change in the pelvic pressure (see Table III).

From the well-known fact that irritation of the urethra and bladder, e.g. in cystoscopy, can produce reflex effects on urinary output (PFLAUMER 1919, BOEMINGHAUS 1924), it does not necessarily follow that ureteric calculi can produce similar reflexes. It should be remembered, however, that the intramural portion of the ureter is actually a part of the bladder, having the same innervation as the latter; from this portion of the ureter reflexes can be set up in the same manner as from other parts of the bladder.

This was noticed distinctly in some of my experiments. As is pointed out in Chapter II, the determination of the pain threshold of the renal pelvis could be facilitated by giving the subject a low caudal anaesthesia; thereby it was possible to avoid disturbances of urinary excretion when carrying out instrumental manipulations in the bladder and ureteric orifices. Where the subjects had received only surface anaesthesia in the urethra, attempts to introduce the somewhat unwieldly balloon catheter into the ureteric orifice often resulted in prolonged ipsilateral anuria. In eleven of these cases, where I succeeded in introducing the catheter without affecting the urinary output, I noted the effect of a more powerful irritation of the intramural portion of the ureter by inflating the balloon in the ureteric orifice, thus testing its sensibility. In eight cases (exps. 16, 18, 19, 24, 28, 31, 44, and 46) the urinary flow ceased instantaneously, while it remained unaffected in the remaining three (exps. 21, 51, and 57). In exps. 19 and 28 splanchnic block was given twenty minutes and ten minutes, respectively, after the onset of anuria. This resulted in an immediate resumption of urinary flow, and repeated distention of the intramural portion of the ureter could thereafter not be made to affect the flow of urine. On the other hand, dilatation of the balloon in other parts of the ureter (5 cm up or more) never produced any noticeable effect on the urinary flow.

It is clear, therefore, that the afferent components of such reflexes from the intramural portion of the ureter are conducted through the sacral nerves, while the efferent components are conducted along nerves which are blocked by splanchnic anaesthesia.

Contralateral disturbances of urinary excretion

Of the various disturbances of urinary output occurring in renal colic, the one that has aroused most interest is *contralateral reflex anuria*. All are agreed that complete bilateral anuria can occur in a unilateral attack of ureteric colic, but there

is difference of opinion as to whether such a phenomenon is necessarily associated with pathological changes in the kidney or urinary tract on the unobstructed side, or whether the function of even a completely normal kidney can be inhibited ("true reflex anuria"). It is unnecessary to review everything that has been written about this problem: it suffices to mention a few important recent publications that include reviews of the older literature in this field.

Among those who have worked on this problem during the last ten years it is only WULFF (1941, 1948) who denies the existence of true contralateral reflex anuria. He bases his belief partly on a large clinical material where many of the patients have undergone intravenous urography during an attack of renal colic, and partly on extensive animal experiments. In WULFF's own material, comprised of more than two thousand cases of renal colic, he did not find a single case of reflex anuria. After a close scrutiny of cases described in the literature as examples of true reflex anuria, he came to the following conclusion:

"A careful analysis of the cases of reflex anuria in the literature has shown that, judged by our present knowledge of urological morbid anatomy and urological diagnostic facilities, most of these cases do not stand this critical examination. Frequently no X-ray examination was performed. Quite a number of cases must be regarded as bilateral ureteral obstruction. Sometimes, for instance, in cases with a fatal outcome, microscopic examination of the so-called sound kidney was omitted. A critical survey of these cases indicates that the possibility of the occurrence of a so-called reflex calculus anuria cannot be completely excluded, but the evidence submitted is hardly conclusive." (1941; auto-quotation 1948, p. 55.)

Since WULLF wrote this the first time, however, further articles have been published by authors who are convinced that reflex anuria can occur in a normal kidney, and they contribute new cases where the phenomenon is presumed to have occurred (LATTIMER 1945, SUERMONDT 1950, ÜBELHÖR 1953). The fact that the renal circulation can be influenced reflexly by various forms of nervous excitation, as shown by TRUETA *et al.* (1947), has lately been used as an argument in favour of the existence of a true reflex anuria (WILDBOLZ 1952).

In his experimental work, WULFF ligated the ureter on one side and studied the effect of this on the urinary output on the contralateral side. In most instances, compensatory polyuria on the unobstructed side resulted fairly rapidly, but in a few cases this polyuria was preceded by pronounced oliguria varying in duration from a few minutes up to over an hour, which was not to be confused with so-called "physiological oliguria", a phenomenon dependent on the reduced total urinary output in both kidneys after the ligation of one ureter. Since anuria did not occur on the contralateral side in a single case, WULFF held that his results argued against the existence of *reno-renal reflexes;* but he omitted to discuss the causation of the transient true contralateral oliguria sometimes occurring after ligation of the ureter.

In my experiments on human subjects I was able to show similar contralateral oliguria upon obstruction of one ureter in a few cases. The details of the experiments

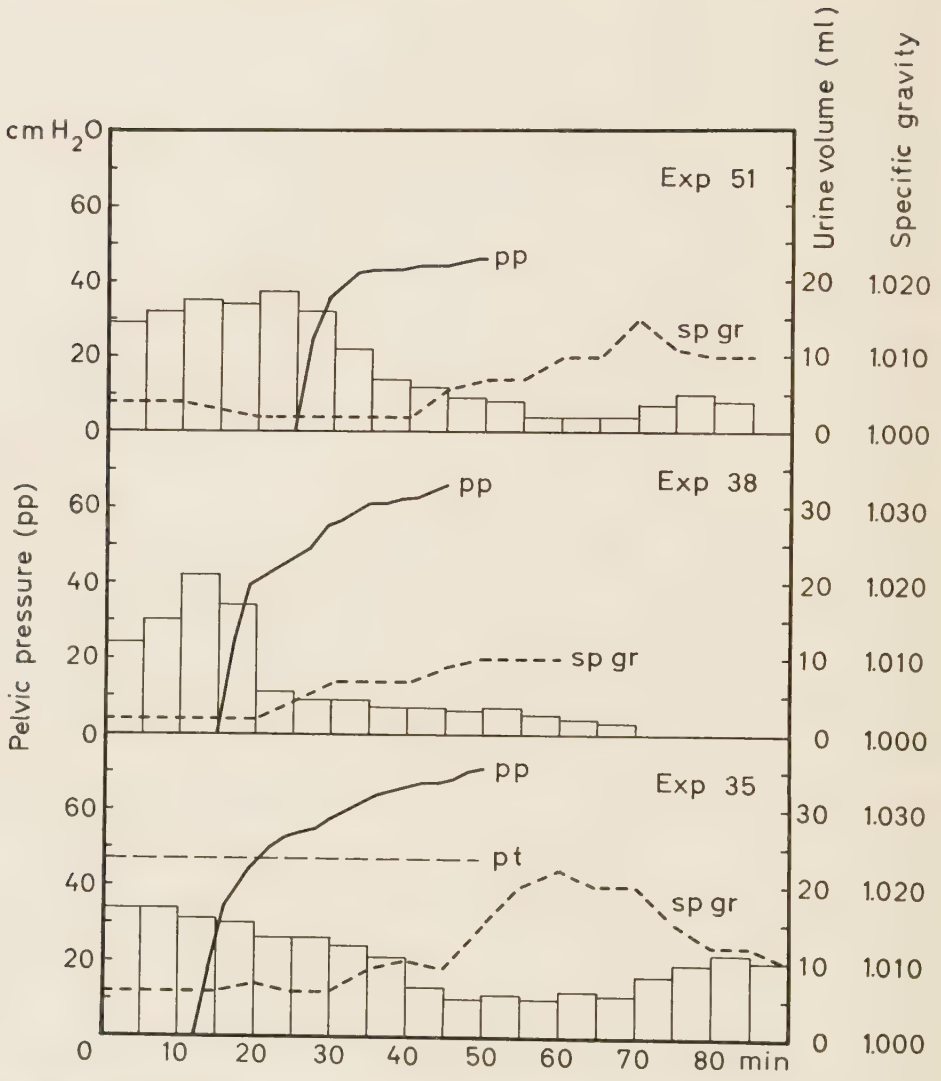

FIG. 18. Diagrams showing the influence of rising pelvic pressure above a ureteric obstruction upon diuresis and specific gravity of the urine on the contralateral side. *pp* = pelvic pressure, *pt* = pain threshold.

were essentially the same as those described in Chapter II. In addition to the catheter equipped with a balloon for obstructing the one ureter, I introduced into the other ureter a catheter, identical to the one described in Chapter X, through which all the urine could be collected without causing any irritation of the ureter once it was in place. In order to maintain a brisk and, as far as possible, a constant flow of urine, the subjects were asked to drink a glass of water (about 200 g) every half hour for

three hours preceding, as well as during, the experiments. The volume and the specific gravity of each five-minute specimen of urine was determined (see Chapter X).

The volumes of urine for several successive five-minute periods having been found to remain stable, I obstructed one ureter with the balloon and noted the rising pressure above the obstruction in the usual way. In three cases out of seven (exps. 35, 38, and 51) I was able to show a marked decrease in the output of urine on the opposite side as the pressure above the obstruction increased, and at the same time an increase in the specific gravity (Fig. 18). In the remaining four cases there were no definite changes in the volume or concentration of the urine during the period in which the ureter was obstructed (50–60 minutes). The subjects in exps. 38 and 51 experienced no pain in connection with the increased pelvic pressure; they had both received an injection of morphine and scopolamine in the usual preoperative dose.

These experiments are too few to permit of any conclusions, but against the background of WULFF's animal experiments they assume some value. It is clear that contralateral oliguria sometimes does occur with unilateral ureteric obstruction, and I find it difficult to explain this on any other basis than a *reno-renal reflex*. Without entering more closely on its mechanism, it seems reasonable to assume the existence of a bilateral vascular reaction of the same nature as that observed unilaterally by BIANCHI and DELL'ADAMI and TREVISINI.

It is clear that *reflex disturbances of urinary output can occur in connection with renal colic*. The exact mechanism of such reflexes is not yet obvious but the phenomenon is probably neurological. A humoral factor may play a certain part, since it has been shown that even a denervated kidney can react to afferent nerve stimuli with inhibited water diuresis (THEOBALD and VERNEY 1935). To explain this, the afferent impulses have been imagined to release antidiuretic hormone from the posterior lobe of the pituitary, thereby influencing tubular absorption.

Whether or not this mechanism plays any part, it is known that afferent and probably also efferent reflex tracts run through the splanchnic nerves. *Splanchnic block has therefore been recommended by many in cases of reflex anuria*, and successful results of this procedure have been noted, not only in cases where the anuria occurred in a diseased kidney, but also in cases where true reflex anuria is claimed to have existed (NEUWIRT 1922, CHAUVIN and BARTHÉLEMY 1943, LUZUY 1950, SUERMONDT 1950). It is obvious that in such a situation the *splanchnic anaesthesia should be given bilaterally.*

Summary

The present work consists of two parts.

The *first part* deals with an experimental investigation intended to contribute to the discussion of the *mechanism of renal colic*. The prevailing confusion on this point seems to arise out of our incomplete knowledge of the sensibility of the upper urinary tract. The few experiments hitherto carried out in this field are incomplete, and usually suffer from the fact that unphysiological methods of stimulation have been used to elicit pain.

In this investigation, made in connection with clinical cystoscopies, it was my object to determine whether pain could be elicited, firstly by local increases in pressure at various points along the ureter, and secondly by obstruction of the ureter, leading to an increase in the urinary pressure within the renal pelvis. The investigation was made with the aid of a ureteric catheter equipped with two lumina, one of which opened onto the tip, and the other into an inflatable rubber balloon. By inflating this balloon it was possible either to apply pressure against the ureteric wall at any point without thereby obstructing the flow of urine, or to obstruct the ureter and record the increasing pressure above the obstruction.

It was found that as high a pressure in the ureter as 150–190 mm Hg did not invariably produce pain; in the lower part of the organ (excluding the intramural portion) it was exceptional to elicit pain in this way, but closer to the renal pelvis the sensibility was greater. In the renal pelvis itself, however, a very moderate rise in pressure (33 mm Hg, on an average) was adequate to produce a pain which in all respects resembled typical renal colic.

In connection with these experiments I made certain observations relevant to peristalsis above an obstruction caused by a balloon in the ureter; these observations lend support to the modern belief that the upper urinary tract displays a state of hypomotility in renal colic. An older conception that hyperperistalsis above an obstruction played a part in the causation of renal colic can, therefore, be rejected, and it would seem to be clear that *the increased pressure in the renal pelvis is the essential cause of the typical pain in renal colic.*

The maximal excretion pressure of the kidney is generally higher than the renal pelvis pressure required for the elicitation of pain. Individual variations in respect

of this may, however, explain the long-debated inconsistencies regarding pain in different types of ureteric obstruction.

The *second part* of the present work deals with the *treatment of renal colic with posterior splanchnic block.*

After a review of the innervation of the upper urinary tract, there follows a discussion on how the pain-conducting nerve fibres can be blocked by means of conduction anaesthesia. Posterior splanchnic block is held to be the most suitable form of conduction anaesthesia for this purpose. The technique, dangers and possible complications are described.

The method was used in fifty-one cases of severe renal colic. *The analgetic effect was invariably complete,* the patients receiving instant relief not only from the pain but also from nausea, meteorism and other disagreeable sensations. As posterior splanchnic block makes it possible to dissipate severe renal colic rapidly and completely without any side-effects whatsoever, *this method is judged to be superior to all other methods of treating this painful condition.*

The possible use of posterior splanchnic block as a diagnostic test in the differential diagnosis of appendicitis and right-sided renal colic was investigated by giving this anaesthesia to a number of patients who were about to be operated for suspected appendicitis (sometimes revealed at operation to be acute enteritis or mesenteric adenitis). In contrast to the cases of renal colic, these patients always retained pain and more especially tenderness, both after unilateral and after bilateral splanchnic block. *The method has thus a certain value as a diagnostic test.*

The effect of splanchnic block on the normal functions of the kidney and the ureter has been discussed, with the object of determining whether descent of a ureteric calculus is influenced with the innervation blocked. It has been shown quite clearly that splanchnic block does not produce any functional disturbances that may be of significance in this respect. On the other hand, *splanchnic block seems to have a favourable effect on the reflex disturbances of renal function—especially reflex anuria—that may occur in association with renal colic.*

References

Arduini, M.: Blocco novocainico dei nervi renali e del plesso ipogastrico. *Urologia 17:* 338, 1950.

Bernard, C.: Leçons sur les propriétés physiologiques et les altérations pathologiques des liquides de l'organisme. Paris 1859.

Bianchi, E. and Dell'Adami, G.: I reflessi vascolari renali dopo legatura ureterale. *Arch. ital. urol. 24:* 325, 1950.

Bieter, R. N.: The effect of the splanchnics upon glomerular blood flow in the frog's kidney. *Am. J. Physiol. 91:* 436, 1930.

Bingham, J. R., Ingelfinger, F. J., and Smithwick, R. H.: The effect of sympathectomy on abdominal pain in man. *Gastroenterology 15:* 18, 1950.

Boeminghaus, H.: Beiträge zur Physiologie der Harnleiter. *Ztschr. f. urol. Chir. 14:* 71, 1924.

Brandesky, W.: Die Beeinflussung der Nieren- und Harnleiterfunktion durch die Paravertebral-Anästhesie. *Ztschr. f. urol. Chir. 39:* 34, 1934 a.

—— Die therapeutische Anwendung der Paravertebral-Anästhesie in der Urologie. *Ztschr. f. urol. Chir. 39:* 82, 1934 b.

Brown, W. E.: Single injection caudal for obstetrical analgesia and the use of pontocaine. *Am. J. Obst. & Gynec. 55:* 717, 1948.

Burton-Opitz, R.: The character of the innervation of the kidney. *Am. J. Physiol. 40:* 437, 1916.

Caldwell, J. M., Marx, H., and Rowntree, L. G.: Renal function after bilateral denervation of the kidneys in normal dogs. *J. Urol. 25:* 351, 1931.

Ceelen, W.: Über "essentielle" Nierenblutungen. *Virchows Arch. f. path. Anat. 275:* 674, 1930.

Chauvin and Barthélemy: Dix nouveaux cas d'anurie traités par l'infiltration anesthésique du pédicule rénal. *J. d'urol. 51:* 98, 1943.

Chesterman, J. T.: Observations on renal pain. *Brit. J. Urol. 17:* 45, 1945.

Davis, L., Pollock, L. J., and Stone, T. T.: Visceral pain. *Surg., Gynec. & Obst. 55: 418*, 1932.

Dempster, W. J. and Joekes, A. M.: Functional studies of the kidney autotransplanted to the neck of dogs. *Acta med. Scandinav. 147:* 99, 1953.

Deuticke, P.: Über die Ureterkolik. *Ztschr. f. Urol. 44:* 124, 1951.

Dourmashkin, R. L.: Dilatation of ureter with rubber bags in the treatment of ureteral calculi. *J. Urol. 15:* 449, 1926.

Engelmann, Th. W.: Zur Physiologie des Ureter. *Arch. f. d. ges. Physiol. 2:* 243, 1869.

Fedoroff, S. P.: Nieren- und Harnleiterchirurgie, 1923. Ref. by Tschernjak, I. S., *Ztschr. f. urol. Chir. 31:* 20, 1931.

Finsterer, H.: Die Bedeutung der Splanchnicus-Anästhesie für die Laparotomie des Oberbauches. *Schmerz 9:* 81, 1936.

Foerster, O.: Die Leitungsbahnen des Schmerzgefühls und die chirurgische Behandlung der Schmerzzustände. *Beitr. z. klin. Chir. Sonderbd. I*, 1927.

Fuchs, F.: Die Hydromechanik der Niere. *Ztschr. f. urol. Chir. 33:* 1, 1931.

Full, H.: Blutdruck und Harnabflussbehinderung. *Berl. klin. Wchnschr. 57:* 1149, 1920.

Gottlieb, R. and Magnus, R.: Über Diurese. *Arch. f. exper. Path. u. Pharmakol. 45:* 248, 1901.

Gottstein, G.: Nephrolithiasis. Lichtenberg, Voelcker & Wildbolz: Handbuch der Urologie, Bd. IV: 272, Berlin 1927.

Grimson, K. S., Hesser, F. H., and Kitchin, W. W.: Early clinical results of transabdominal celiac and superior mesenteric ganglionectomy, vagotomy, or transthoracic splanchnicectomy in patients with chronic abdominal visceral pain. *Surgery 22:* 230, 1947.

Gruber, Ch. M.: The autonomic innervation of the genito-urinary system. *Physiol. Rev. 13:* 497, 1933.

Gubergritz, M. M. and Istschenko, I. N.: Zur Frage der Entstehung der Schmerzempfindungen in den Nieren. *Ztschr. f. d. ges. exper. Med. 52:* 619, 1926.

Guyon, F.: Influence de la tension intra-rénale sur les fonctions du rein. *Compt. rend. Acad. d. sc. 114:* 457, 1892.

Heidenhain, R.: Handbuch der Physiologie, herausgeg. v. Hermann, Bd. V, 1: 325, Leipzig 1883.

Henderson, V. E.: The factors of the ureter pressure. *J. Physiol. 33:* 175, 1905.

Hermann, M.: Über den Einfluss des Blutdruckes auf die Secretion des Harns. *Sitzungsb. d. K. Akad. d. Wissensch. Math.-naturw. Cl. 45:* 317, 1861.

Hertz, A. F.: The sensibility of the alimentary canal in health and disease. *Lancet 89, 1:* 1051, 1119, 1187, 1911.

Hinman, F. and Lee-Brown, R. K.: Pyelovenous back flow. *J.A.M.A. 82:* 607, 1924.

Hoffman, S. J.: Ureteric calculi. *South African M. J. 24:* 425, 1950.

Hollinshead, W. H.: Anatomic pathways for pain from the upper part of the abdomen. *Proc. Staff Meet., Mayo Clin. 28:* 385, 1953.

Hortolomei, N., Streya, M., and Burghele, Th.: Über Hämaturien bei Nierenbeckenentleerungsstörungen. *Ztschr. f. urol. Chir. 41:* 234, 1935.

Huber, A.: Recherches physiologiques sur la résorption rénale. Thése, Paris 1895.

Huggins, Ch. and Scott, W. W.: Cutaneous ureterostomy with contra-lateral ureteral ligation. *J. Urol. 53:* 325, 1945.

Hultén, O.: Über Bauchschmerz und dorsale Splanchnikusanästhesie. *Acta chir. Scandinav. 85:* 281, 1941.

Kappis, M.: Erfahrungen mit Lokalanästhesie bei Bauchoperation. *Verhandl. d. deutsch. Gesellsch. f. Chir. 43:* 87, 1914.

—— Sensibilität und lokale Anästhesie im chirurgischen Gebiet der Bauchhöhle mit besonderer Berücksichtigung der Splanchnicusanästhesie. *Beitr. z. klin. Chir. 115:* 161, 1919.

— -- Zur Technik der Splanchnicusanästhesie. *Zentralbl. f. Chir. 47:* 98, 1920.

Kappis, M. and Gerlach, F.: Die differentialdiagnostische Bedeutung der paravertebralen Novokaineinspritzung. *Med. klin. 19:* 1184, 1923.

Kellgren, J. H.: On the distribution of pain arising from deep somatic structures with charts of segmental pain areas. *Clin. Sc. 4:* 35, 1939.

Klumpp, K.: Die konservative Behandlung der akuten Pankreasnekrose durch Novocainblockade. *Zentralbl, f. Chir. 77:* 1249, 1952.

Köhler, R.: Investigations on backflow in retrograde pyelography. *Acta radiol. suppl. 99*, 1953.

KULENKAMPFF, D.: Die Verwendung der Kappis'schen Splanchnicusanästhesie zur pathogenetischen und diagnostischen Klärung der Erkrankungen des Oberbauches. *Zentralbl. f. Chir. 50:* 208, 1923.

LABAT, G.: Regional anaesthesia, with special reference to "splanchnic analgesia". *Brit. J. Surg. 8:* 278, 1920.

LAPIDES, J.: The physiology of the intact human ureter. *J. Urol. 59:* 501, 1948.

LATTIMER, J. K.: A plan for the management of anuria. *J. Urol. 54:* 312, 1945.

LÄWEN, A.: Paravertebrale Novokaininjektionen zur Differentialdiagnose intraabdomineller Erkrankungen. *Zentralbl. f. Chir. 49:* 1510, 1922 a.

—— Über segmentäre Schmerzaufhebung durch paravertebrale Novokaininjektionen zur Differentialdiagnose intraabdominaler Erkrankungen. *München. med. Wchnschr. 69, 2:* 1423, 1922 b.

—— Weitere Erfahrungen über paravertebrale Schmerzaufhebung zur Differentialdiagnose von Erkrankungen der Gallenblase, des Magens, der Niere und des Wurmfortsatzes sowie zur Behandlung postoperativer Lungenkomplikationen. *Zentralbl. f. Chir. 50:* 461, 1923.

—— Weitere Beiträge zur Sacralanästhesie und zur Paravertebralanästhesie für diagnostische und operative Zwecke. *München. med. Wchnschr. 72, 2:* 1449, 1925.

LEARMONTH, J. R.: Neurosurgery in the treatment of diseases of the urinary bladder. *J. Urol. 26:* 13, 1931.

LENNANDER, K. G.: Beobachtungen über die Sensibilität in der Bauchhöhle. *Mitt. a. d. Grenzgeb. d. Med. u. Chir. 10:* 38, 1902.

—— Über lokale Anästhesie und über Sensibilität in Organ und Gewebe, weitere Beobachtungen II. *Mitt. a. d. Grenzgeb. d. Med. u. Chir. 15:* 465, 1906 a.

—— Leibschmerzen, ein Versuch, einige von ihnen zu erklären. *Mitt. a. d. Grenzgeb. d. Med. u. Chir. 16: 24,* 1906 b.

LEWIS, TH.: Pain. New York 1943.

LICHTENAUER, F.: Experimentelle Untersuchungen zur Kenntnis der Nierenbecken- und Harnleitererweiterung. *Arch. f. klin. Chir. 260:* 34, 1948.

LION, K.: Über den Einfluss paravertebraler Injektionen auf die Nierentätigkeit. *Med. Klin. 23, 1:* 15, 1927.

LOBENHOFFER, W.: Funktionsprüfungen an transplantierten Nieren. *Mitt. a. d. Grenzgeb. d. Med. u. Chir. 26:* 197, 1913.

LUZUY, M.: Les infiltrations du sympathique: Physiologie, indications, techniques. Paris 1950.

MCCAHEY, J. F.: Renal and ureteral responses to obstruction. *J. Urol. 63:* 976, 1950.

MACINTOSH, R. R. and BRYCE-SMITH, R.: Local analgesia: Abdominal surgery. Edinburgh and London 1953.

MACLEAN, J. T., CARROLL, J. J., and GRAVES, H. B.: The treatment of ureteral colic and ureteral calculus by thoraco-lumbar sympathetic block: Preliminary report. *J. Urol. 61:* 204, 1949.

MCLELLAN, A. M. and GOODELL, H.: Pain from the bladder, ureter and kidney pelvis. *Research Publ., A. Nerv. & Ment. Dis. 23:* 252, 1943.

MANDL, F.: Blockade und Chirurgie des Sympathicus. Wien 1953.

MASON, J. M.: Dangers attending injections of the kidney pelvis for pyelography. *J.A.M.A. 62:* 839, 1914.

MITCHELL, G. A. G.: The innervation of the kidney, ureter, testicle and epididymis. *J. Anat. 70:* 10, 1935.

- The renal nerves. *Brit. J. Urol. 22:* 269, 1950.

NAEGELI, TH.: Splanchnicusanästhesie bei chirurgischen Eingriffen und schmerzhaften Affektionen in der Bauchhöhle. *Zentralbl. f. Chir. 46:* 749, 1919.

NARATH, P.: Renal pelvis and ureter. New York 1951.

NEUWIRT, K.: Ein Beitrag zur Therapie der Reflexanurie. *Ztschr. f. urol. Chir. 11: 75,* 1922.

OCKERBLAD, N. F. and CARLSON, H. E.: Distribution of ureteral pain. *J. Urol. 39:* 745, 1938.

OLSSON, O.: Studies on back-flow in excretion urography. *Acta radiol. suppl. 70,* 1948.

PAL, J.: Die krampflösende Wirkung der paravertebralen Injektion. *Wien. klin. Wchnschr. 37:* 1323, 1924.

PAPIN, E.: Étude sur la douleur rénale. *Arch. d. mal. d. reins 4:* 1, 253, 385, 1929.

PFEIFER, W.: Grundlagen der funktionellen Röntgendiagnostik. Stuttgart 1949.

PFLAUMER, E.: Cystoskopische Beobachtungen zur Physiologie der Harnleiter und Nieren. *Ztschr. f. Urol. 13:* 367, 1919.

—— Der Ureterstein, seine Diagnose und Behandlung. *München. med. Wchnschr. 77, 2:* 1675, 1717, 1930.

PITKIN, G. P.: Conduction Anesthesia. Philadelphia 1946.

QUINBY, W. C.: The function of the kidney when deprived of its nerves. *J. Exper. Med. 23:* 535, 1916.

RAY, B. S. and NEILL, CH. L.: Abdominal visceral sensation in man. *Ann. Surg. 126:* 709, 1947.

REXED, B.: Spinalnervrötternas durarotfickor: anatomi, histologi, patologi. *Nord. med. 41:* 726, 1949.

RHOADS, C. P., VAN SLYKE, D. D., HILLER, A., and ALVING, A. S.: The effects of novocainization and total section of the nerves of the renal pedicle on renal blood flow and function. *Am. J. Physiol. 110:* 392, 1934.

RISHOLM, L.: Conduction anaesthesia in obstetrics. *Acta Soc. Med. Upsal. 55:* 323, 1950.

ROSS, J. A.: Studies in pyelorenal backflow. *Brit. J. Urol. 24:* 27, 1952.

SCHRAGER, V. L. and IVY, A. C.: Symptoms produced by distention of the gallbladder and biliary ducts. *Surg., Gynec. & Obst. 47:* 1, 1928.

SMITH, H. W.: The Kidney. New York 1951.

DE SOUSA PEREIRA, A.: Blocking of the splanchnic nerves and the first lumbar sympathetic ganglion. *Arch. Surg. 53:* 32, 1946.

STARLING, E. H.: The glomerular functions of the kidney. *J. Physiol. 24:* 317, 1899.

SUERMONDT, W. F.: Reflex anuria. *Arch. Chir. Neerland. 1:* 269, 1950.

DE TAKATS, G.: Splanchnic anaesthesia. *Surg., Gynec. & Obst. 44:* 501, 1927.

DE TAKATS, G. and WALTER, L. E.: The treatment of pancreatic pain by splanchnic nerve section. *Surg., Gynec. & Obst. 85:* 742, 1947.

DE TAKATS, G., WALTER, L. E., and LASNER, J.: Splanchnic nerve section for pancreatic pain. *Ann. Surg. 131:* 44, 1950.

THEOBALD, G. W. and VERNEY, E. B.: The mechanical factors which affect the secretion of urine in mammals, and their operation during pregnancy. *Quart. J. Exper. Physiol. 25:* 341, 1935.

TRATTNER, H. R.: Graphic registration of the function of the human ureter with the hydrophorograph. *J. Urol. 28:* 1, 1932.

TREVISINI, A.: Trattamento di 133 casi di calcolosi ureterale con infiltrazione novocainica del pedunculo renale. *Urologia 18:* 430, 1951.

—— Il comportamento arteriografico del rene durante la colica nell'uomo. *Urologia 19:* 139, 1952.

TRUETA, J., BARCLAY, A. E., DANIEL, P. M., FRANKLIN, K. J., and PRICHARD, M. M. L.: Studies of the renal circulation. Oxford 1947.

Übelhör, R.: Bemerkungen zur reflektorischen Anurie. *Ztschr. f. Urol. 46:* 19, 1953.

Watel, J.: L'Innervation de l'uretère. Thèse, Lille 1941.

Weens, H. S. and Florence, Th. J.: Nephrography. *Am. J. Roentgenol. 57:* 338, 1947.

Wharton, L. R.: The innervation of the ureter, with respect to denervation. *J. Urol. 28:* 639, 1932.

White, J. C., Smithwick, R. H., and Simeone, F. A.: The autonomic nervous system. Third edition. New York 1952.

Wildbolz, H.: Lehrbuch der Urologie. Berlin 1952.

Winton, F. R.: The relation between diuresis and maximum ureter pressure, and its bearing on current methods of estimating the rate of glomerular filtration. *J. Physiol. 83:* 38 P, 1934.

—— Physical factors involved in the activities of the mammalian kidney. *Physiol. Rev. 17:* 408, 1937.

Wulff, H. B.: Über die sog. reflektorische Steinanurie. Lund 1941.

—— Urinary excretion in acute unilateral renal and ureteral blocking. Lund 1948.

Wüllenweber, G.: Untersuchungen über die Druckverhältnisse in Blase, Ureter und Nierenbecken (besonders während der Schwangerschaft). *Deutsches Arch. klin. Med. 165:* 296, 1929.

Uppsala 1954. Almqvist & Wiksells Boktryckeri AB 547415